TRANSACTIONS

OF

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SIXTY-SIXTH ANNUAL MEETING

James R. Gudger, M. D.
Editor

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Transactions of The Association of Life Insurance Medical Directors of America

SIXTY-SIXTH ANNUAL MEETING

The Sixty-sixth Annual Meeting of The Association of Life Insurance Medical Directors of America convened on Wednesday morning, October 23, 1957, in the Skytop Room of the Hotel Statler, New York, N. Y., at ten fifty-five o'clock, President Edson E. Getman, M. D., presiding.

PRESIDENT GETMAN — We are pleased to have with us Dr. John E. Boland, Chairman of the Medical Section of the American Life Convention, who will address us concerning the next meeting of that organization.

DR. JOHN E. BOLAND — Mr. President, fellow members and guests! It affords me much pleasure to bring to you greetings from the Medical Section of the American Life Convention, and I am pleased to have this opportunity to extend to each of you a cordial invitation to attend the meeting which will be held at the Broadmoor Hotel, Colorado Springs, Colorado, Monday, Tuesday and Wednesday — June 9, 10 and 11. The Broadmoor is known the world over for its scenic natural beauty and is indeed the garden spot of the West. Dr. William H. Scoins is our Program Chairman and something new has been added. I feel sure it will prove interesting and educational, particularly from an underwriting standpoint. May I suggest that you register for the meeting no later than Sunday, June 8, as we plan to start promptly on schedule Monday morning. We will devote the afternoons to golf, tennis, riding, etc. The Broadmoor boasts of an 18 hole championship golf course. For those preferring not to scale the Rocky Mountains, electric carts are available. Dr. Francis Bicknell,

our golf chairman, has arranged a 36 hole tournament. Dr. Howard McCue is tennis chairman. Prizes will be awarded, as usual. Bring your wives. Provision has been made for their entertainment.

PRESIDENT GETMAN — It is always a great pleasure to welcome our esteemed friend and fellow member, Dr. Francis R. Dieuaide, Scientific Director of the Life Insurance Medical Research Fund.

DR. DIEUAIDE — In the past year, contributions to the Fund enabled us to award grants and fellowships amounting to $1,061,357 — a total exceeding one million dollars for the first time. Administrative expenses came to $67,237, considerably less than seven per cent of our receipts for the year.

The Fund is currently aiding some 115 research programs, in addition to supporting 22 research fellowships. Investigators working with the Fund's aid last year published their results in some two hundred scientific articles. Their reports to the Fund, as well as these publications, show that many helpful contributions have been made toward better understanding and management of cardiovascular diseases. Many new findings have been made in regard to the role of fats in the diet, their absorption, and their metabolism in the body. This new knowledge is highly provocative as to further work bearing on the causes of arteriosclerosis. While it is not yet possible to make a rational plan for the prevention of arteriosclerosis, it is reasonable to expect a solution of the problem.

Experimental results show that several different procedures involving dietary management, hormonal or drug treatment, and others, are capable of influencing the development and progress of arteriosclerosis.

More attention has been paid to the rôle of the arterial wall itself. At least after the sclerotic process starts it is not passive, for sizable amounts of phospholipid are then synthesized within the arterial wall, as well as cholesterol.

Recent work has borne more closely on thrombosis, without which arteriosclerosis may be a minor ailment. Some results tend to indicate a relation between dietary composition and intravascular blood coagulability. The striking action of the lipemia-clearing

factor system is the basis of promising work. A striking discovery is that high fat meals can cause heart pain, whereas clearing the blood of fat gives relief.

Current studies bearing on the mechanism of hypertension lend emphasis to the rôle of sodium and potassium metabolism. The amounts and especially the ratios of these minerals are clearly important. Key processes may be hormonal actions and kidney function.

At present, the prevalent conception is that chronic hypertensive disease has a great many different causes. This is perhaps the result of our ignorance, rather than our insight. In the meantime, it is gratifying that the management of hypertensive patients has been much improved, especially in severe cases.

An extracellular streptococcal product has been found, minute quantities of which can quickly impair the heart's ability to contract. This discovery may help explain the relation of streptococci to rheumatic heart disease.

Open heart surgery has recently made great strides forward. In one clinic aided by the Fund postoperative deaths have fallen to a rate of 2.5 per cent as the result of improved apparatus and technic. Current work is directed toward applications in acquired heart disease, including coronary occlusion.

Our understanding of the conditions for efficient and regular heart action has been much improved through new information about the metabolism of heart muscle and the control of heart rhythm. Prevention or control of heart failure and arrhythmias has thus been enhanced.

The Fund's awards have aided research in 120 institutions widely distributed throughout the United States and Canada, with a very few abroad. A total of 228 individuals have received fellowships. Both grants and fellowships are much sought after and highly valued, as shown by the large number of excellent applications, both for original awards and for renewals. The work of the Fund is now widely known and appreciated.

As administrator of the Fund, I wish to express my great appreciation for the essential aid of the members of the Advisory

Council and especially of the Medical Directors' Representatives, including Dr. Ylvisaker and Dr. Gordon, the appointees of the Association. Their constant readiness to give advice and help means everything to us.

Our new Annual Report has just been distributed. If any member of the Association failed to receive his copy, a postcard addressed to me will bring one by return mail. We believe you will all be interested in reading the concise summary provided of last year's work.

PRESIDENT GETMAN — Thank you, Dr. Dieuaide.

Due to the amount of literature being put forth, both in newspapers and in the medical press, regarding the dangers of excessive radiation, your Program Committee felt that it would be appropriate to have some part of our program devoted to that subject.

Dr. Reynold F. Brown, Chief Medical Examiner in San Francisco for the New York Life Insurance Company, addressed us in 1953 on the status of radiation safety at that time. Since that date, he has been made a member of the Advisory Committee on Isotope Distribution to the Atomic Energy Commission. Dr. Brown has consented to bring us some of the more up-to-date thinking and ideas in regard to radiation safety.

THE STATUS OF RADIATION SAFETY IN 1957

REYNOLD F. BROWN, M. D.*
Chief Medical Examiner
New York Life Insurance Company
San Francisco

Four years ago, when you gave me the opportunity to discuss the status of radiation safety, the general problem revolved around the question of whether or not it was probable that the increase in man-made ionizing radiation of all types would remain within the range of maximum permissible exposures. Our conclusions then that these assumptions were valid remain unaltered today.

However, in the intervening four years a new question has been raised. Within the presently conceived limits of maximum permissible exposure is there harm of a biological nature and, if so, to what extent is the harm caused by ionizing radiation? So much publicity has been given to this problem in scientific literature and other media that it would be redundant to review the claims and counterclaims on this subject. But, it may be profitable to review some of the factual material in order to evaluate the substance behind the two major opposing viewpoints. One group states that any radiation is harmful, even the low levels within the range of maximum permissible exposure being *significantly harmful,* that the present level should be greatly reduced and further steps taken to keep the range of controlled ionizing radiation as near zero as possible. The other viewpoint is that the range of ionizing radiation exposure to which man is *presently* exposed is of such an amount that *no significant* biological harm will ensue and the present course may be continued, the maximum permissible exposures being reconsidered from time to time and adjusted in what appears to be the best interests of man at the particular time of the review. The literature furnishes some factual data that will help appraise the present status of radiation safety.

There is considerable material on the biological effect from the

*Member, Advisory Committee on Isotope Distribution to the Atomic Energy Commission.

delivery of hundreds of roentgens to man and animals in various periods of time, but as the dose is gradually reduced it enters an area where there is very little factual material. On the low end of the exposure scale there are measurements made on what might be called the natural background of ionizing radiation from various sources. Taking this as the minimum to which man is exposed, one may try to determine the biological effect as the dose is gradually increased, thus establishing a range within which the so-called safe limits may lie. No matter which end of the scale is pursued an unknown area is encountered, and it is precisely the one on which information is needed. Because the high range data is not within the present subject let us begin with the factual material which has been measured to represent the low end of the exposure scale.

Figure 1 is a measure of the cosmic radiation and the local gamma rays emitted by naturally radioactive materials in our environment. The measurements were made at various locations ranging from approximately sea level to high altitudes, such as Pike's Peak. While the amounts registered here are not large, the concept that the range of the natural background is wide is important in the argument that the present range of ionizing radiation is acceptable. The wide variations of exposure at various elevations suggest that man apparently can live satisfactorily within a considerable range of ionizing radiation without any detectable biologically disastrous effect, and this includes genetic and somatic effects. Therefore, it is probable that a slight increase within this broad range would scarcely affect the biological balance.

The bottom line represents man-made fallout contamination. It is drawn as a straight line showing that relative to the natural background man-made radiation from fallout has been very small. However, it is misleading in that the background contributed by the atom bombs is actually irregular, and thus similar to the natural background because the fallout is dependent upon variable weather conditions and altitude, so that the deposits are unevenly distributed. It is only the probable average increase that is envisioned here. To complete this picture, the addition of another hundred milliroentgens per year would represent the average per capita

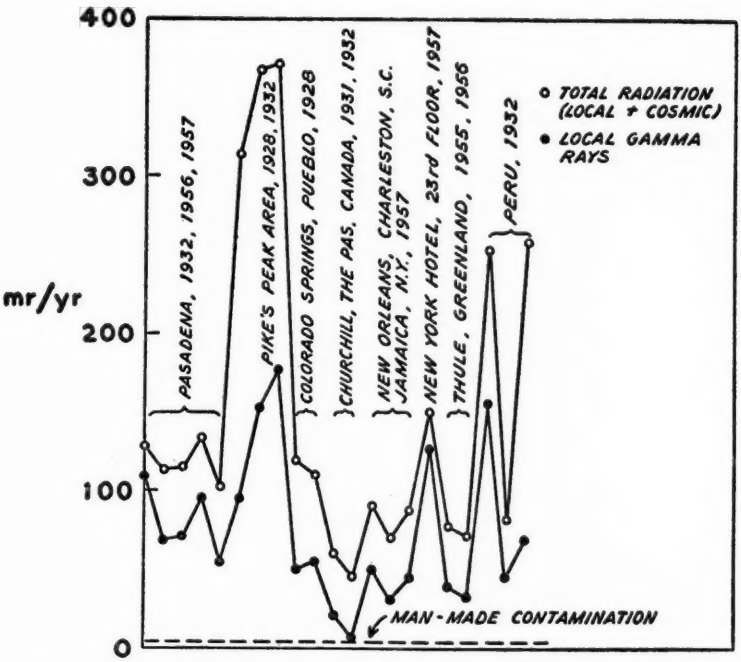

Fig. 1. The range of background radiation. (Reprinted from Neher, H. V.: Gamma rays from local radioactive sources, Science. **125**:1088 [May 31] 1957.) Reproduced by courtesy of the Editor.

contribution below age 30 from medical use of ionizing radiation to the population of the United States. This is the estimate from the National Academy of Sciences' report to which further reference will be made later. Thus, when the increase from the medical use of radiation is superimposed on natural background, it is of such magnitude that the natural variations make unlikely any experiment of the direct observation type that will differentiate the biological effect of either one. If this is the case, then on what factual basis can it be determined that even this range of exposure is biologically harmful and possibly biologically significantly harmful? One of the most sensitive biological indicators in the hands of investigators has been the genetic effect of ionizing radiation. It is a well-established fact that with high levels of exposure to

biological preparations there is an irrefutable genetic effect and if these effects are plotted as the dosage is reduced, the line, if extended, appears to pass through zero or very close to zero (figure 2). The number of points which have been determined along this line are grouped together at rather high dosages. When one begins to examine the material in the lower dosage range to

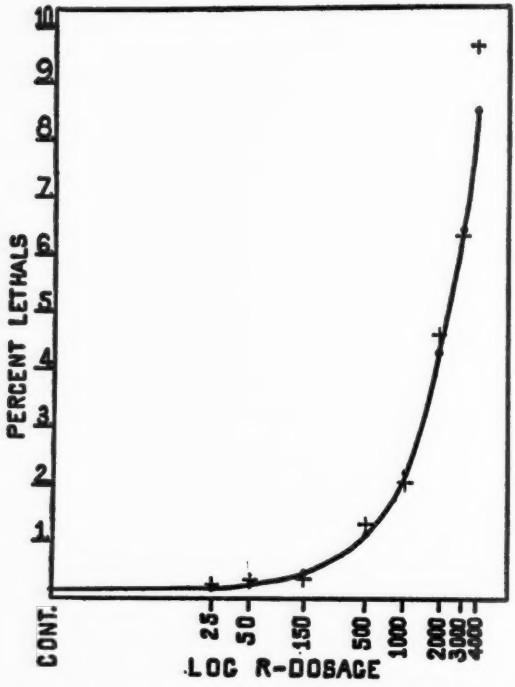

Fig. 2. Mutation rate vs. dose in Drosophila. (Reprinted from Spencer, W. P. and Stern, C.: Experiments to test validity of linear R-dosage mutation frequency relation in Drosophila at low dosage, Genetics. **33**:43, 1948.) Reproduced by courtesy of the Editor.

see whether or not any points far down the curve can be established, then difficulties of interpretation are encountered. This particular group of experiments, commonly referred to as factual data, attempts to show that low levels of ionizing radiation are biologically effective and table 1 summarizes five of these ex-

TABLE 1

Mutation Rate in Drosophila at Low Dosage*

Treatment	No. of Controls	No. of Experimentals	Mutation rate percent		
			Controls	Experimentals	Diff.
50 r, 2.3-5 minutes exposure, not aged (Spencer and Stern)	73,901	31,560	0.0974	0.2440	0.1466
52.5 r, 21 days exposure, aged (Caspari and Stern)	56,252	51,963	0.2489	0.2848	0.0359
50 r, 24 hours exposure after 20 days aging	44,601	46,232	0.1682	0.2834	0.1152
100 r, 21 days exposure, aged	22,958	31,562	0.2352	0.4658	0.2306
52.5 r, 21 days exposure, aged	36,184	29,424	0.1765	0.2542	0.0777

*Reprinted from Uphoff, D. E. and Stern, C.: Genetic effects of low intensity irradiation, Science. **109**:609–610 (June 17) 1949. Reproduced by courtesy of the Editor.

periments. In the first experiment on Drosophila it was found that the mutation rate following 50 roentgens showed an increase of 14 per cent over the control. When this experiment was repeated using sperm that had been aged for 21 days it was found that the increase in mutation rate was only three per cent and this was not statistically significant. Since this raised the point of why these observations were in conflict, the other groups of experiments were performed. Fifty roentgens were given again to aged sperm and this time there was a difference of 11 per cent which was statistically significant. Then the dosage was doubled and the effect was approximately doubled. The 50 roentgen exposure was repeated in a similar manner, but not exactly the same, and again it resulted in a seven per cent difference. Now, if one examines these individually as we have done, it is found that the majority of them support the original idea that the mutation rate was proportional to the x-ray dose. Notice, however, by examining the control spontaneous mutation rate in the total group of experiments, a wide range of variability in the spontaneous mutation rate is observed and that it is purely fortuitous (based upon the spontaneous mutation rate) when the different results become significant. The actual exposure following radiation is quite consistent; it is only the spontaneous mutation rate that is changed. As a matter of fact, one might say that the ionizing radiation had a stabilizing effect on the mutation rate as it is observed here. This, as a group picture, is not very strong evidence that a critical point has been completely established in the curve at 50 roentgens. It would appear difficult at this level to stabilize the spontaneous mutation rate to such a degree that the effect of the x-rays could be observed. When the amount of radiation is reduced, the effects get smaller and the experiments get larger in order to get a statistically significant result until, finally, at levels much lower than these one is faced with an overwhelming size to the experiment which then becomes impractical to handle. Therefore, some extrapolation becomes almost a necessity. It is this fundamental fact, complicating the observations on the effect of small amounts of ionizing radiation, that has made it impractical to do any direct human studies; and it is also the reason that the study on the Japanese survivors has been inconclusive as to any genetic effect.

Now, there is another side to this argument. Observers cannot

extend the experimental evidence in animals below even 100 roentgens, which is the zone of importance when considering the cumulative effect genetically of ionizing radiation. If one accepts the idea that any amount of ionizing radiation applied to the population produces its random quota of genetic effect, the rate of exposure makes no difference. When X roentgens to the preparation has accumulated it will have the same genetic effect as if it had been given all at once, as long as this is received during the reproductive lifetime of the population under study. Therefore, the geneticists add together all of the radiation exposure received by man over his 30 years of life. This is accepted as the average age at which reproduction is completed and is used as a total amount of radiation received, correlating it as much as possible with the experimental results (in which the radiation was delivered in a short period of time) and which are subject to the difficulties of interpretation just discussed. It should be emphasized that the acceptability of this segment of the argument rests strongly upon the premise of no-threshhold effect. Further, in this no-threshhold effect is the supposition that the ionizing events which cause the genetic mutation are single random events on an all-or-none basis, and that they are irreparable and irreversible. This is, in effect, the so-called target theory of the biological effect of ionizing radiation. The workers who are studying this experimental data continue to seek in man a detectable effect at low dosages, such as life shortening or incidence of disease, which would support the supposition and theory of no-threshhold and establish definitely the importance of single ionizing events in producing disease or harmful effects on man.

On the average, the total amount of ionizing radiation received up to age 30 from all sources is lower than any *direct* experimental evidence that has been shown to produce significant damage, genetic or otherwise. There is one type of material in the literature which is an exception to the above statement and that is the purely mathematical manipulation of results similar to the experiments just described and extrapolating results to theoretical low levels, but these manipulations are still tenuous in their arguments so that more acceptable evidence is needed to establish their validity. They do not warrant a secure place in the structure of the accepted concept of maximum permissible exposure at this

time. If more convincing evidence is produced in the future, it may very well be necessary to revise the working rules presently accepted as reasonable.

In the discussion of figure 1 the average exposure to the population under age 30 was shown to be in the region of approximately 100 milliroentgens per year from medical use of ionizing radiation which in 30 years would be the 3000 milliroentgens, or approximately the three to four roentgens estimated in the National Academy of Sciences' report. We might increase our understanding of the particular subject by examining in greater detail the medical use of ionizing radiation, especially that for diagnostic purposes. There are approximately eighty-one and a half million people in the United States (table 2) under age 30 and this is the group of people on whom it is necessary to figure the total genetically potentially harmful exposure. The examinations of diagnostic x-ray can be broken down by the percentage of examinations done according to the procedures listed in table 3. It is evident that chest examinations constitute about 33 per cent of the total group as might be expected; stomach, duodenum, esophagus around 12 per cent; abdomen 6; spine 6; colon 5½; skull 4½ per cent; and they drop off very rapidly down the list until there is quite a sizable group that range between one and two per cent of the examinations. Now, as might be expected, the groups that deliver the greatest amount of radiation to the gonads are those examinations where the gonads come in the direct beam of the x-ray so that to understand the distribution of the radiation from medical use one must study the type of examinations done and the exposure received from the particular examination (table 3).

Table 4 shows the radiography done by radiologists and all physicians in the adult group, ages 12 to 30, in the categories shown. Columns five and six list the total number of roentgens delivered to the gonads of the entire population within this age group from these examinations. It should be noted that column four gives the percentage of examinations in the total group and that in some fields (of 12, 13 and 10 per cent) there are values ranging from 350,000 to over a million roentgens, depending upon the area examined. The total exposure is, therefore, more

TABLE 2

Population of United States According to Age and Sex in 1954*

	Total	Male	Percent	Female	Percent
Total	162,414,000	80,696,000	49.7%	81,718,000	50.3%
Population under 30 years	81,696,000	41,384,000	50.5%	40,312,000	49.5%
Percentage of total under 30	50.3%	51.1%		49.4%	
Population under 12 years	40,112,000	20,481,000	51.0%	19,631,000	49.0%
Percentage of total under 12	24.7%	25.3%		24.0%	
Population between 12 and 30	41,584,000	20,903,000		20,681,000	
Percentage of total	25.6%	25.8%		25.4%	
Total in 1949	149,215,000				

Rate of increase of total population between 1950 and 1955 has averaged 2.7 million per year.

*Reprinted from Laughlin, J. S. and Pullman, I.: Preliminary Report of Gonadal Dose Received in Medical Use of X-rays. Prepared for the Genetics Panel of the National Academy of Sciences' Study of the Biological Effects of Atomic Radiation. (November 19) 1956. Reproduced by courtesy of the Author.

TABLE 3

Roentgenographic Examinations by Conditions*

	Per Cent
Chest	33.01
Stomach, duodenum, esophagus	12.22
Abdomen	6.47
Spine	6.34
Colon	5.66
Skull orbits, sella turcica	4.42
Pelvis and hip joints	4.16
Thigh, leg, knee	3.17
Urographic excretion	2.82
Urinary tract	2.60
Heart	2.08
Gallbladder	1.98
Ankle and foot	1.85
Sinuses, paranasal and mastoids	1.80
Wrist and hand	1.58
Arm, forearm, elbow	1.38
Urographic, retrograde	1.32
Sacroiliac	1.27
Shoulder girdle	1.15
Facial bones	0.77
Cholangiogram	0.59
Ribs and sternum	0.58
Bronchogram	0.48
Ventriculogram	0.47
Small intestine	0.44
Encephalogram	0.38
Miscellaneous	1.00
Total number of examinations 30,355	100.00

*Reprinted from Donaldson, S. W.: The practice of radiology in the United States: Facts and figures, Am. J. Roentgenol. 66:929–946, 1951. Reproduced by courtesy of the Editor.

directly related to the area examined than to the number of examinations. For instance, of the nine million examinations listed under the adult category, chest x-rays accounted for 35 per cent of the total as seen in item five. However, the total gonadal exposure in this age group was 2500 roentgens. This, as compared to the total of three million roentgens (total radiography), brings into sharp focus the relatively insignificant contribution from chest x-rays and that in rounding off evenly this section of diagnostic x-ray procedures this amount could easily be disregarded. Therefore, if all chest x-ray examinations were to be discontinued it would have very little effect on the gonadal exposure of the population. *This brings into good perspective the hysteria that has been prevalent in a good many pieces of literature on the dangers from certain diagnostic examinations.* It further illustrates the impossibility of broad generalized indictments of misuse.

Table 5 shows another segment of this total picture, namely the obstetrical examinations. Here, attention is focused particularly upon pelvimetries and examination of the pelvis of pregnant women. In considering these figures it may be better to begin at the bottom with the line "Total Diagnostic Gonadal Dose", and this is eleven million roentgens. These total figures were taken from the over-all use of diagnostic x-ray, some of which have not been considered at this time. The line above that indicates the total obstetrical dose to be almost two million roentgens so that this would approximate 18 to 20 per cent of the total gonadal exposure, and it is received from obstetrical type examinations which constitute less than one per cent of the radiological examinations. Discontinuation of obstetrical radiology would significantly reduce the population exposures but the effect of this would have to be balanced against the expected increase in morbidity and mortality in obstetrics if the films were omitted. It might be possible to restrict the indications for obstetrical radiology to those conditions of greater necessity than may at the present time be in use and thus reduce the over-all total exposure without affecting the practice of obstetrics to a great degree. However, all of these possibilities are based upon conjecture. The original figures, shown here as compiled by Laughlin and Pullman, determined that 23 per cent of primiparas had a pelvi-

TABLE 4

Probable Annual Genetically Effective Medical Diagnostic X-ray Dose to the Gonads of the Population in the United States*

A. General Radiography

(1) Type and Number of Examinations	(2) Gonad Dose/Exam. Milliroentgens Female	(3) Gonad Dose/Exam. Milliroentgens Male	(4) Percent of Exams in Group	(5) Total Number of Roentgens Per Year Female	(6) Total Number of Roentgens Per Year Male
Radiography (Radiologists and all physicians)					
Adults (age 12-30)					
4.4 million exams — female					
4.9 " " — male					
1. **Genito-urinary tract**					
a. Pyelography	1,200	2,000	4.1	215,000	405,000
b. Urinary tract	1,000	300	2.6	113,000	38,000
c. Salpingography	1,000	—	0.3	130,000	—
2. **Gastrointestinal tract**					
a. Abdomen and colon	500	200	12	262,000	118,000
b. Stomach and upper GI	300	200	13	170,000	128,000
c. Gall bladder	200	10	3	26,000	150,000
3. **Skeleton-pelvic region**	1,000	2,000	10	440,000	980,000
4. **Skeleton-extremities and chest**	0.5	1.0	12	300	600
5. **Chest**	0.3	1.2	35	500	2,000
6. **Head**	0.2	0.6	8	70	200
Total radiography — adults				1,357,000	1,687,000

B. Obstetrical Examinations

(1) Type and Number of Examinations	(2) Gonad Dose/Exam. Milliroentgens Female	(3) Gonad Dose/Exam. Milliroentgens Male	(4) Percent of Exams in Group	(5) Total Number of Roentgens Per Year Female	(6) Total Number of Roentgens Per Year Male
Obstetrical Examinations					
A. Mothers					
0.3 million effective pelvimetries	2,500	—	100	750,000	—
2.5 million effective births for					
1. Placentography (1 film per exam)	260	—	0.6	3,900	—
2. Abdomen flat plate (1 film)	260	—	3.4	23,000	—
3. Chest (1 film/exam)	0.1	—	47.4	100	—
Total obstetrical — mothers				776,000	
B. Fetus					
3.6 million births in hospitals (50% male, 50% female)					
1. Pelvimetry	4,000	4,000	11.4	546,000**	546,000
2. Abdomen flat plate (1 film)	400	400	3.4	16,500	16,500
3. Placentography (1 film)	400	400	0.6	2,900	2,900
4. Chest (1 film)	0.3	0.3	47.4	200	200
Total obstetrical — fetus				566,000	566,000
Total Obstetrical Dose				1,341,600	566,000
Total Diagnostic Gonad Dose				5,200,000	5,800,000

**All fetus doses have been reallocated to correspond statistically with the other examinations. The multiplying factor is 0.67.

*Reprinted from Laughlin, J. S. and Pullman, I.: Preliminary Report of Gonadal Dose Received in Medical Use of X-rays. Prepared for the Genetics Panel of the National Academy of Sciences' Study of the Biological Effects of Atomic Radiation. (November 19) 1956. Reproduced by courtesy of the Author.

metry during their pregnancy. These figures were compiled in the New York area and applied to the population as a whole. Due to the significant volume represented by them in the total exposure, this field may deserve further exploration to determine whether these assumptions and samplings are indeed correct when applied to the total population of the United States. However, accepting them at their face value it might be useful to approach the question as to whether or not 23 per cent of primiparas really need a pelvimetry. Certainly, 23 per cent of women do not have abnormal pelves nor are the fetal pelvic proportions in 23 per cent of primiparas abnormal. But, it is likewise true that in order to diagnose correctly the percentage that shows disproportion it is necessary to examine a larger group in order to identify the really significant ones, and it may very well be that 23 per cent is a figure that cannot be lowered to any real degree without affecting the morbidity and mortality, and that the medical reward far outweighs the gonadal exposure hazard. Even though 23 per cent appears high in relation to the total amount of radiation, it may very well be one of the most useful segments of the gonadal exposure.

These possibilities are mentioned in order to show the need for a program of constant appraisal and investigation, and to point out directly that there is need for further study of the subject and that *there is time for study.*

These x-ray exposures from medical use can be modified at any time. They do not have the irreversible status that long-lived isotopes have from fallout contamination and they will remain under constant appraisal as they have been for the past 50 years. One reference to the fact that the medical profession has been aware of danger in its use of x-ray is to be found in a textbook on x-ray diagnosis published in 1912 by Bythell and Barclay. Its closing paragraph includes the statement that stress must be laid once more upon the importance of avoiding all unnecessary exposure to x-rays. And it is further stated, *"It is advisable to have some idea of the exposure we may give the patient during the course of an x-ray examination without causing him to suffer any ill effects."* In a textbook by McKee on x-rays and radium treatment published in 1921, a section on the biological explanation

of the effect of x-rays contains a paragraph devoted to the known *histogenetic* phenomena even though it was admitted that the morphological picture failed to explain the fundamental changes. The author referred to the work of Hertwig who had demonstrated that the visibly damaged cell structures were the chromosomes. They deduced that the chromatin was directly injured by radiation, that it was the primary injury and all subsequent developments were consequent to it.

These extracts are from books used by the medical profession and indicate that published information has not been disregarded by its members, nor are they unaware of its potential danger. Their further awareness has been evidenced by the fact that for 25 years or more there have been committees, whose organization and establishment were supported by the medical profession, that have devoted their interest and time in evaluating the literature of the world on this subject. They have constantly reappraised the use of ionizing radiation and continually lowered the maximum permissible exposure. These are continuing bodies, constantly at work with their representation on an international basis. At any time they can be called into session to study new material and to publish recommendations for change. They are constantly active in this field and are establishing new study groups for new problems and will examine any material brought to their attention that may be of significance in relation to the general problem. Charges that the medical profession has been blind to the dangers of ionizing radiation, or that they should now institute a program of limiting the use of ionizing radiation are, therefore, unfounded. They have had such a program in charge of proper committees on a national and international basis for several decades. As just mentioned, the statements from an ordinary textbook of 1912 reveal a very accurate concept, even in the light of present knowledge, of the danger in the use of ionizing radiation. It is really some of the other disciplines of science who are the newcomers to the field of danger, and while they are welcome allies in the study they are by no means the originators of the concept of danger. In the field of the medical use of ionizing radiation we can be assured that the dosage will continue to decrease due to increased efficiency of mechanical generators, new equipment, better technics, and perhaps even more rigidly selected indications for x-ray

examinations, if such be needed. In the field of fallout contamination, this is a global problem that has political implications and is beyond the ability of the organizations mentioned to modify, and as such is somewhat beyond the limits of present discussion. These remarks serve only to point out some of the reasoning behind the argument pertaining to the relative safety of the present exposures as measured at this time. The main purpose, however, is to discuss some factual material by which to appraise the literature as it will continue to appear in the coming months and years.

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PRESIDENT GETMAN — Thank you very much, Dr. Brown. I have asked Dr. Charles M. Barrett, who is Professor of Radiation Therapy of the Department of Surgery and Radiology at the University of Cincinnati, and Medical Director of the Western and Southern Life Insurance Company of Cincinnati, and who has had a wide experience in this field, to discuss Dr. Brown's paper.

DR. CHARLES M. BARRETT — Mr. Chairman, I was indeed pleased to be asked to discuss Dr. Brown's paper, but I was much more pleased after I read it and saw the quality of a really first rate presentation.

This paper has impressed me deeply because it has accomplished something needed for a long time. What it has done is to present a rather objective view of a problem which is emotionally charged by both sides of the argument as extremely exciting and subject to a great deal of controversy. I certainly intend to refer to it a great many times in the future.

A few remarks may be in order about some of the practical

problems in regard to the genetic effects of radiation and the question of whether or not the population is getting too much x-ray, the safety of physicians and the safety of patients.

First, I would like to point out that it is very difficult, as Dr. Brown indicated, to transmit adequately experimental evidence from mice or from the fruit fly to humans. It can be done on an extrapolation basis, but it is difficult to do it practically. Those of you who have worked in the animal laboratory know how difficult it is to transmit such experiments to humans.

As far as atomic radiation is concerned, there is very little that can be done about that. It is entirely a political problem, and, as the newspapers have indicated, we do not seem to have complete control as to whether or not there is going to be an increase in the background of radiation.

One important practical point is that fluoroscopy has been eliminated. This is one instance where patients in the young age group, children, and those sensitive to radiation, are liable to be exposed, and some damage may be compiled over a long period of time. We have recommended to our pediatricians that they not fluoroscope the child every time he comes to the office.

The problem of the pediatrician who fluoroscopes the child at every opportunity has existed in our locality for some time. We feel that this is probably not a good thing for the children and, certainly, it has not been a good idea for the pediatricians, because I personally know one who has developed leukemia, which, I am certain, was due to over-irradiation.

Reducing the amount of fluoroscopy is also important. The use of spot films gives less dose to the gonads, less general body irradiation, and the spot film device is very practical and can eliminate extensive and prolonged fluoroscopy.

We have also added something as far as the protection of the gonads is concerned. We are now in the position of not knowing exactly how much damage takes place, and whether or not the charges made by the geneticists are true. We recognize that, scientifically, from the experimental point of view, there have been some recognizable mutations that have occurred as a result of radiation. We recognize that chemicals, heat, and other forms

of stimulus also can produce mutations. But until we know more about it and have reached more valid conclusions, we have decided to protect the gonads of people, when possible, during routine examinations.

The use of new equipment is important. The image amplifier, which was adapted by Russell, Morgan and Chamberlin, has been of tremendous help in limiting the amount of radiation received by the patient, and the radiologist as well, while making fluoroscopic examinations. It goes without saying that proper measurement of the radiation received by the patient and the radiologist is extremely important for him to know. You, as doctors who may use fluoroscopy in your practice or in examination of people at the insurance company's home office, must realize that it is of the utmost importance that you be protected. You are just as vulnerable as the radiologist to radiation, and you must have recent equipment, of proper survey, and know exactly how much your particular machine puts out, and what the scatter radiation from the patient and from the table is to you. That is quite important. Many people, particularly in internal medicine, seem to feel a certain immunity toward radiation because they use it casually. This is, of course, not true at all and can result in very serious consequences.

I should also like to mention one other item of extreme importance — the reduction of radiation therapy in children. One of the first remarkable effects that we were able to note — and this was ten years or so ago — was that in children who had been treated for enlarged thymus, there was an increase in the development of thyroid cancer. At about the age of 30 or 35 or 40, there was a definite increase in the amount of thyroid cancer in those individuals who had received radiation therapy as children for enlarged thymus.

At the present time, we are conducting a study in regard to treatment of lymphadenitis in children, trying to determine whether or not there has been an increase in the amount of hemopoietic disease as contrasted with control groups.

In conclusion, therefore, I should like to point out that this problem is a controversial one, and it is good that we can have

heated arguments as to what the dangers and hazards are by stimulated proponents. It has been advantageous for us as radiologists to have outside influences exerted. I also think that it is extremely important and valuable for us to have had the opinions of individuals such as Dr. Brown, who take a completely objective point of view, and try to evaluate this in its proper place.

Radiation is going to continue as an extremely valuable tool, in therapy and in diagnosis; and we will continue to protect the people and the doctors. There is no question about it being shelved as a result of a sort of genocide. With proper protection, and proper adaptation of the principles now being established, we will arrive at a very safe conclusion in regard to x-rays, both in diagnosis and therapy.

PRESIDENT GETMAN — Thank you, Dr. Barrett. Our next paper is "Hematologic Disorders: Their Disabilities and Prognoses".

Hematology has grown so extensively over the past few years that it has become increasingly difficult for all of us to keep abreast of even a fraction of the newer concepts in diagnosis, treatment, and prognosis. Dr. Paul Reznikoff, Professor of Clinical Medicine at the Cornell University Medical College, has been a leader and has had a vast experience in this field. I know that you are all eager to hear his presentation.

HEMATOLOGIC DISORDERS: THEIR DISABILITIES AND PROGNOSES

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When a patient consults a physician about his illness, the first thought which occurs to the doctor, as a rule, is the cause of the disorder and then he directs his attention to therapy. However, there are other features about disease which are of importance, especially among those conditions for which the cause is unknown and where there may be no available treatment. This refers particularly to disability and prognosis.

Suppose a patient has an abnormality. What about it? Is he disabled, and, if so, how? What is his outlook? Can he live to a ripe old age, or will the disability probably shorten his life?

A text for my remarks might be: What profiteth the patient if his blood picture is abnormal but he feels well and can carry on his work and live reasonably happily? The answer is obviously that he profiteth a great deal.

It is realized that we may have a conflict of interests in considering this subject. The practicing physician is concerned primarily with his individual patient. You are too; but you also deal with statistics. You want to know what is going to happen to a hundred or a thousand individuals who have certain deviations from the normal. However, the problems of disability in relation to work and of prognosis with respect to span of life are of considerable importance in your special medical field.

In hematology we deal with (1) the formed elements of the blood — red blood cells, white blood cells and thrombocytes or platelets; (2) the substances concerned with clotting; and (3) the specific bodies which are important not only in medical problems, such as transfusions, but also are of considerable significance in the fields of anthropology and medical jurisprudence.

It is not possible to review all the hematologic problems seen

in practice, but a few have been selected which are significant as far as disability and prognosis are concerned.

First, let us consider the anemias. Before listing the main causes of anemia, it should be realized that anemia is not a disease but is a sign of a disease. All anemias are secondary to something. This is not always appreciated by physicians who often treat anemia without trying to determine the etiology of this state.

The chief causes of anemia are: (1) hemorrhage; (2) deficiency of necessary elements which make up hemoglobin and red blood cells, such as iron, the maturative factors like vitamin B_{12} and folic acid, proteins, ascorbic acid and thyroid substance; (3) hemolysis which may be due to intrinsic abnormalities of the red blood cells, as in familial spherocytic jaundice, Mediterranean anemia and sickle cell anemia; or due to certain extrinsic factors, as in hemolytic infections, chronic infections like tuberculosis, malaria, acute lead poisoning, neoplasms, lymphomas; or in the state known as hypersplenism characterized by the formation of autoagglutinins demonstrated often by a positive Coombs' test; (4) depression of the bone marrow (mistakenly called aplastic anemia since in most cases all the blood elements are involved) due to agents like benzol, irradiation, chloramphenicol, also seen in leukemia and in many cases of unknown cause; and (5) metabolic disturbances interfering with red blood cell and hemoglobin formation as in uremia.

It must be remembered that many patients have multiple causes for anemia. For example, in pernicious anemia the red blood cell maturation is defective and these cells also break up easily. In leukemia the erythrogenous elements of the bone marrow are encroached upon by the white blood cell proliferation, but a hemolytic factor also contributes to the anemia.

Let us now consider the disability and prognosis in these anemias. In the first place it is necessary to emphasize the fact that in most cases the disability is due principally to the condition causing the anemia, although the anemia may contribute to the symptoms. For anemia itself to produce conspicuous disability, the decrease in red blood cells and hemoglobin must be of such a degree or must be produced so rapidly that the patient is suffer-

ing from anoxia. Unless the deficiency in the oxygen-carrying function of the blood causes lack of oxygen in the tissues, the patient will not suffer much disability from the anemia itself. The term "tired blood" is ridiculous. The young girl who stands so pathetically against the wall may be menstruating excessively, or is not eating adequately, and may have some decrease in her blood count. But she rarely is anoxic.

A patient can lie in bed with a fairly pronounced anemia and be perfectly comfortable. If he must perform some physical activity, with the same blood count, then he will be distressed. It is just a question of anoxia rather than some absolute figure.

We must now turn to specific anemias. What is the outlook for the bleeding patient? That depends upon whether the bleeding can be stopped. If he is bleeding from hemorrhoids and they are excised, the prognosis is excellent. The same is often true of bleeding from uterine fibroids. However, if the bleeding is due to a duodenal ulcer the problem may be more difficult to solve, but such patients are cured by medical or surgical treatment. Suppose the hemorrhage is due to esophageal varices, secondary to cirrhosis of the liver, and because of this condition the patient also has an increased prothrombin time and perhaps a low platelet count, then we are confronted with a much more difficult situation.

Therefore, the prognosis in a patient who is bleeding depends upon the rapidity of blood loss, how much blood he has lost and the site of the hemorrhage — whether the bleeding can be terminated.

Suppose the patient has anemia because of defective nutrition. If he absorbs iron and protein normally, and receives the proper nourishment, then his outlook is good. However, if he is a poverty-stricken inhabitant of a country like India, or if a woman persists in ingesting a defective, fadist diet, then the patient will probably continue to be anemic. Until recently the anemia of sprue was very difficult to treat because of faulty absorption due to the diseased small intestine. Now with a gluten-free diet these patients may be returned to a normal state.

I now wish to discuss two type of anemia which are frequently misjudged as far as disability and prognosis are concerned. A

patient with pernicious anemia who is treated adequately, and has no neurological symptoms or signs, and no disability, is a perfectly normal individual as far as life span is concerned. The only exception to this statement is the fact that the incidence of carcinoma of the stomach is greater in these otherwise normal persons than in people of the same age group, but possibly not any higher incidence than in others of the same age group with achlorhydria.

Another person who may have a great deal of difficulty in obtaining a position or normal insurance rates after a blood count is performed is the young woman who has mild thalassemia or the Mediterranean trait. Her hemoglobin value is low but she feels well and often does not know about her abnormality. Her only concern should be not to marry a man with the same trait or their children may have severe Cooley's anemia. But people with the Mediterranean trait have no disabilities and their span of life is a normal one.

As far as hemolytic anemias are concerned, the most dramatic success in therapy is the almost 100 per cent cure rate in patients with spherocytic jaundice by splenectomy. They have no disabilities and their prognosis is excellent after the spleen is removed. Unfortunately, this is not true in the acquired type. Steroids help in some of these individuals but often they must be given transfusions to keep them comfortable and the prognosis is not good.

The anemia due to bone marrow depression is a serious disease and the outlook is usually not good regardless of the cause. As mentioned previously, these patients with hypoplastic marrows as a rule also have depression of the platelets and white blood cells.

We might now consider the condition which is the opposite of anemia, that is, polycythemia vera. As you know, this disorder has three main characteristics, namely, erythrocytosis, splenomegaly and cyanosis. Some authorities look upon polycythemia as a serious disease but those who have followed these patients for many years think of it as a chronic condition and that both disability and prognosis depend upon the complications. It is not uncommon to see polycythemic patients live for ten or fifteen years after the diagnosis is made. Since most patients are in their fifties when polycythemia is first noted, one cannot consider the outlook

as being too gloomy, especially when compared to many other diseases which occur at this time of life.

What are the disabling complications of polycythemia? These patients frequently have thrombosis, probably due to the increased viscosity of the blood, but also because of vascular changes. Paradoxically, they tend to bleed easily. We do not know the reason for this but it is dangerous to operate upon a polycythemic patient until his hematocrit value is 50 per cent or lower. These individuals have the following complications — cirrhosis, gout, hypertension which accompanies the disease or develops during the course of the illness, and in a number the terminal condition is leukemia or hypoplastic marrow. It is generally believed that irradiation therapy is probably not the cause of the terminal leukemia since a similar percentage of untreated patients and those treated by phlebotomies also finally die of leukemia. Sometimes the patient's chief disability is the mechanical inconvenience of the large spleen which is due not only to the erythrocytosis but also to extramedullary hematopoiesis. In general, most of these patients may be free of severe disability for many years and if their blood counts can be kept down to a normal level by P_{32} or phlebotomies, they live a fairly useful life.

Hemorrhagic states are due to many causes. Some authorities divide bleeding conditions into two classes — those due to quantitative platelet deficiency or thrombocytopenia, and those that occur in the presence of a normal platelet count or non-thrombocytopenic conditions. Idiopathic thrombocytopenic purpura is helped by steroid therapy and is cured in about 70 per cent of all cases by splenectomy. There are many causes of thrombocytopenic purpura. Some of these are drugs like sulfonamides, quinidine and also irradiation. Also, some individuals susceptible to hair dyes containing paraphenylendiamine develop thrombocytopenia. In some diseases platelet depression occurs. Examples of these are acute leukemia, hypoplastic bone marrow, cirrhosis, Gaucher's disease, lupus erythematosus, and a rare and fatal condition called thrombotic thrombocytopenic purpura characterized by a marked decrease in platelets, hemorrhages, fever, jaundice and sporadic and migratory neurological signs.

There are many non-thrombocytopenic purpuric states. Some

are due to infections, such as subacute bacterial endocarditis, familial telangiectasia, and scurvy. These patients have normal platelet counts. Aspirin may cause gastric bleeding in certain individuals. Senile purpura occurs with atrophy of the skin. A very common type of purpura is due to fragile vessels. This often occurs in women and may be familial. This condition is unpleasant for cosmetic reasons but these patients rarely have serious bleeding. Bleeding may be due to the lack of one of the coagulation factors. In hemophilia the patient has inherited a deficiency of antihemophilic globulin. Other factors which may be lacking, or abnormally decreased, are prothrombin, plasma thromboplastin antecedent, plasma thromboplastin component, a labile factor, a stable factor and fibrinogen. Hemorrhage may also be due to the presence of an anticoagulation factor. Fibrinogen deficiency may be congenital or acquired as in premature separation of the placenta with amniotic embolization. Also, some patients have a qualitative platelet abnormality called thrombasthenia.

It is obvious that in hemorrhagic conditions the disability and prognosis depend upon the cause of the bleeding. For example, hemophilic patients can be helped considerably by fresh blood transfusions and antihemophilic factor when bleeding or before an operation or tooth extraction. The prognosis of course remains unfavorable. In idiopathic thrombocytopenic purpura, as mentioned previously, splenectomy may cure the majority of patients. But some of these who are apparently normal immediately after the spleen is removed, relapse and this is not due in most cases to accessory spleens missed by the surgeon. On the other hand, patients are seen with no increase in platelets following splenectomy and for some reason they do not bleed any more. The use of vitamin K in hypoprothrombinemia, and fibrinogen administration in patients with fibrinogenopenia may be life saving.

Therefore, in hemorrhagic states the disability is obviously due to bleeding. If the cause of the bleeding is a transitory one, or can be combated by some therapeutic procedure, then the prognosis is good.

Leukemia is the greatest enemy of the hematologist. It is increasing in frequency and as the late Sir Lionel Whitby stated

we have not made appreciable progress in prolonging the life of these patients. However, it must be remembered that leukemia varies tremendously in disability and prognosis in different patients. Some patients with acute leukemia die within a few months following the first evidences of the disease or even sooner, and some with chronic leukemia live a normal life and die of old age, such as those with benign lymphocytic leukemia. Patients with acute leukemia — acute in every sense of the word, including fever, thrombocytopenia, leukopenia, anemia and bone marrow crowded with blasts — are known to have had a remission and live fairly active lives for a few years. These, of course, are exceptions. Some of my patients have lived five and ten years following a diagnosis of acute leukemia. However, one cannot foretell when a leukemic person will suddenly develop a non-reversible relapse.

What are the main disabilities due to leukemia? They are anemia, bleeding and fever. As a rule enlargement of the spleen, liver and lymph nodes is not disabling and intercurrent infection, while always mentioned in textbooks, is not in my experience especially frequent or severe. The fever in leukemia is usually due to the disease itself rather than to infections.

The important fact to remember is that disability and prognosis in leukemia depend upon the individual case and one cannot be certain of what will happen to a specific patient. The course may actually change from day to day. One of the worst disappointments a physician experiences is to have a patient, who apparently is progressing quite well, suddenly become desperately ill with a striking relapse.

Some other hematologic disorders might be mentioned. Neutropenia, generally called agranulocytosis, has a much better prognosis than it had previously because of the use of steroids and antibiotics like penicillin. The disease is usually acute and the disability is marked.

Infectious mononucleosis is a fairly benign disorder. A few deaths have been reported. However, infectious mononucleosis may cause lack of energy and easy fatigability for a long time. Many of the patients also have abnormal liver function tests and some observers believe that disability may be correlated better

with liver damage than with an abnormal blood count or a positive heterophil agglutination test.

We are recognizing an increasing number of persons with multiple myeloma. Certainly the majority of these patients are disabled, principally due to pain. Many of them have anemia, and some have thrombocytopenia. I do not know whether urethane helps these individuals as Rundles believes. Some of the patients with multiple myeloma have cryoglobulin in the blood plasma which precipitates under cold conditions and results in purpura. Many with localized bone lesions experience considerable relief with local irradiation.

How then can one assay a patient's disability and prognosis in a hematologic condition or, for that matter, in any other disease? Most important, of course, is to determine the cause of the illness. However, what is actually disabling the patient are his complaints and his limitation of activity.

There are men who can spend their entire day as an observer in front of a television set but cannot help wipe dishes, even when seated. They are disabled for useful work. There are women who can sit at a bridge table all day, but suffer disability when they must cook. A physician must gauge that type of disability by his knowledge of human nature. But, by and large, a patient's disability depends upon his complaints and his inability to carry on his usual functions.

Then, the question arises whether the patient's disease will be responsible for a shortened life. As mentioned previously one must individualize. If pernicious anemia is treated, he may live a normal span of years provided he does not develop carcinoma of the stomach. If he has benign lymphocytic leukemia, I consider him a good risk. If a patient has thalassemia minor she is, in my opinion, perfectly normal. Therefore, in hematologic disorders the patient provides the chief answer about disability and prognosis and the blood picture is of secondary but of considerable importance.

PRESIDENT GETMAN — Thank you very much indeed, Dr. Reznikoff. I am sure that we were all stimulated by your presentation.

I should like to ask Dr. J. Randolph Beard of the Mutual

Benefit Life Insurance Company of Newark if he would like to discuss Dr. Reznikoff's paper.

DR. J. RANDOLPH BEARD — We are all privileged to have had an outstanding authority in the field of hematology present many phases of this interesting subject in a most comprehensive and understanding way. I am sure that all of my colleagues join me in commending Dr. Reznikoff on his very fine presentation.

My first acquaintance with Dr. Reznikoff was some thirty years ago, at which time he was listing among his many interests that of agranulocytosis, particularly in relation to the introduction of pyramidon or amidopyrine at that time. After pyramidon more or less left the market, the incidence of agranulocytosis varied with the various new drugs that had been introduced into medicine.

From an insurance standpoint, we, like many of the physicians, become enthusiastic whenever a new drug appears to be a panacea for one of the impairments which we are considering. We liberalize our underwriting and very often do not give much thought to the side effects which may take place as a result of some of these newer drugs.

My thoughts turn primarily to such impairments that we now accept while under treatment, namely, epilepsy, arterial hypertension, and, possibly in the future, diabetes, treated by oral medication.

Dr. Reznikoff also mentioned pernicious anemia. In the light of modern therapy, this is possibly a misnomer, for it is no longer pernicious. From an underwriting standpoint, there has been considerable liberalization during the past ten years in considering applicants with hypertension for insurance. Here, again, we may pause to think of other impairments which may occur with pernicious anemia. Bethall and Harrington report gallbladder dysfunction anywhere from 22.5 per cent to 42 per cent in these cases. More specifically, cholecystitis and cholelithiasis have been found in about 16 per cent of them. Carcinoma of the stomach is greater among persons with pernicious anemia, and the work of Kaplan and Rigor at the University of Minnesota brings out the fact that it occurs about three times as often as it does in a control group. This was primarily based on autopsy

figures; but they also did a study on living individuals, and x-rays revealed carcinoma in about 6.6 per cent of the individuals.

We do not have an opportunity to follow the health of our policyholders once the insurance is issued. The chief dangers in pernicious anemia lie in inadequate dosage, intercurrent infection interfering with the utilization of specific antianemic agents, and complications arising from neurological involvement.

This is a chronic disease, and many patients are reluctant to continue treatment for long periods of time or for life. Relapses often follow the onset of the intercurrent disease, or the need for surgery often leads to neglect in the antianemic therapy at a time when it is most needed.

Often, we have applicants with a diagnosis of polycythemia. Just as often, it is difficult to obtain supporting information. There are three classifications: relative, transient, and absolute. The terms, "erythrocytosis", denoting polycythemia which occurs in response to some unknown stimulus, and "erythremia", a disease of unknown etiology, are coming into more common use when referring to the two forms of polycythemia.

Erythrocytosis is often associated with congenital heart disease or chronic pulmonary conditions, chronic mountain sickness, and in relation to chemical and physical agents.

Erythremia, which we know as polycythemia vera, is a disease of insidious onset, chronic course, and unknown etiology. This type of case may have a duration of ten to fifteen years, but is usually shorter because of intercurrent infections. The individual with polycythemia vera is not insurable, except for a mortality ratio well beyond the usual. Other types of polycythemia should be placed in a risk classification according to cause.

Rarely in life insurance underwriting are we faced with a decision in cases of leukemia. I refer primarily to the chronic lymphocytic and myelogenous leukemias in particular. We have all reviewed death claims in which we felt that the onset of the difficulty was prior to the underwriting. Perhaps these cases were in a period of remission or aleukemic leukemia. In the accident and health phase of our business, problems may be presented by all the various types of leukemias.

In idiopathic thrombocytopenic purpura, we have been relatively liberal in the underwriting where splenectomy has been the choice of treatment. One should bear in mind, however, that this procedure is not a panacea.

A report by Lozman of 303 cases of purpura following splenectomy revealed that 61 per cent had complete remission, 17 per cent incomplete, and 22 per cent failures. Consideration of the medically treated cases of idiopathic purpura should be on the basis of a high mortality experience, if acceptable at all. The figures mentioned in the cases following operation would be reversed, namely, about 70 per cent or more falling in the poor risk category.

The subject of Dr. Reznikoff's presentation is of interest to all of us. From a life standpoint, even though we are in a risk business, many of these conditions would not be acceptable, or, if they are, at a very high rating. From the health and accident standpoint, however, there are many of these conditions that could be considered, and those are of importance to all here. The hematologist, of course, has an opportunity to follow his patients. In our office, we speak of ourselves as "one-shot diagnosticians". We have to make up our minds; the papers are signed, and that is the end of it as far as we are concerned. With that in mind, I think, we should proceed with some caution.

PRESIDENT GETMAN — Thank you, Dr. Beard.

Our next paper should be of the greatest interest to all of us, not only in the insurance field, but also to clinicians at large. It is entitled, "Diagnosis, Prognosis, and Treatment of Hypertensive Cardiovascular Disease".

We are, indeed, fortunate in having a speaker who has done and is continuing to do such an enormous amount of work in this field. I am sure you all know that he was instrumental in introducing into this country Rauwolfia as a hypotensive agent and, only within the past few days, has made a still further promising discovery in this field. Since 1953, he has served as vice president and president-elect of the American Heart Association, and next week will assume the presidency, in Chicago. As a matter of fact, Dr. Wilkins tells me that he must leave immediately

after this presentation, since he has to be in Chicago for the current meeting of the Association.

I take great pleasure in presenting to you Dr. Robert W. Wilkins, Professor of Medicine, Boston University School of Medicine.

DIAGNOSIS, PROGNOSIS AND TREATMENT OF HYPERTENSIVE CARDIOVASCULAR DISEASE

ROBERT W. WILKINS, M. D.
Professor of Medicine
Boston University School of Medicine

Obviously, the etiological classification of a disease can be approached from many points of view. The types of cases discussed here are divided as seen clinically, realizing that sometimes a clear separation between types is impossible. It is well known that the so-called essential hypertensive group is the largest, and when patients ask what is meant by "essential", I frequently reply, "Well, that means we know essentially nothing about it." It is the great mysterious group, probably constituting 90 per cent of the cases seen.

Proceeding to the further classification we are more and more frequently able to diagnose renal hypertension. This can be done now by differential studies on the two kidneys. Thus, it is possible to prove that unilateral kidney disease and sometimes to suspect that bilateral kidney disease, arising in such primary diseases as glomerulonephritis or particularly pyelonephritis, is the cause of the hypertension.

Rarer types include those arising from abnormalities of the adrenal gland, one of these being, of course, pheochromocytoma. A great deal has been written recently about primary aldosteronism, produced by cortical adenomas of the adrenal gland which secrete excessive amounts of aldosterone. There are seen in practice also a few cases of Cushing's syndrome with hypertension, and rarely, a case of so-called adult adrenogenital disease which may have hypertension as one feature.

Rarer still are the cases of neurogenic hypertension. One of these is Cushing's reflex hypertension which he produced experimentally in animals by raising the intraspinal pressure and intracranial pressure. Hypertension, sometimes of very startling and severe degree, is observed in such diseases as encephalitis, poliomyelitis, myelopathies, and ordinary neuropathies. Again, the

clinician must always remember coarctation of the aorta and should palpate the femoral arteries to be sure that he is not dealing with hypertension in the upper extremities alone.

Finally, there is toxemia of pregnancy, rarely seen, but important in the hypertension group.

This type of etiological classification must be borne in mind when a new patient is seen and diagnostic studies are undertaken. Accurate measurement of blood pressure is the easiest, simplest, and most readily repeated of all the procedures in the diagnostic survey. As mentioned, we are also interested in the pulses, particularly in the femoral arteries. Of course, we do complete blood examinations as a routine procedure. Likewise, in our studies on the urine the fractional phenolsulfonphthalein test is now stressed. In this, specimens are collected at 15 minutes, half an hour, one hour and two hours after the injection of the dye. This is a particularly useful test of kidney function, since we believe that the kidneys should excrete 25 per cent of the injected dye within the first 15 minutes.

Obviously, x-rays and electrocardiograms are also important, particularly to determine the degree of left ventricular hypertrophy of the heart.

Hypertension can be further classified into two broad groups. We would prefer not to use the term "malignant", but because of its general acceptance its use is continued while endeavoring to modify it slowly into the terms "accelerated phase" or "accelerated form" of hypertension. If untreated the disease is usually fatal within two years. The type following a more gradual course, also called benign, usually continues longer than ten years, the average being 20 years. However, the average age at onset is 32 and the average age at death is 52 years. Thus, life expectancy is reduced by about 20 years in persons having this disease.

One can further divide these cases as to severity by the complications that are usually considered to be the result of long-continued high blood pressure. These are: vascular disease as revealed by examination of the ocular fundi, examination of the heart, especially through use of the electrocardiogram, x-ray evidence of involvement of the aorta, and palpable evidence of in-

volvement of the major arteries. We also rely on the familiar clinical picture of small or large strokes and evidences of damage to the kidneys.

Therefore, when a new patient is seen there is quite an elaborate program of diagnostic procedures that may have to be done; and it is not possible to review here the detailed differential tests that sometimes have to be used to make a diagnosis in some of the rarer forms.

What is done with this information once it is obtained? The treatment is based upon it and, also, the prognosis depends upon it. One should remember that the functions of the physician include not only diagnosis and treatment but also prognosis for the benefit of the patient himself and his family. This very important function of the physician is sometimes neglected. In some aspects this may involve the "art" of medicine. Every patient is different and one must arrive at a summary on the basis of the available information of all the factors for or against a longer or shorter outlook in a given case.

It can be stated unequivocably, however, that renal impairment with uremia is a very bad prognostic sign. Cardiac impairment, especially if it includes coronary insufficiency and marked enlargement, while also serious, is not nearly as poor a prognostic sign as is renal insufficiency. The same may be said of strokes or cerebrovascular disturbances. Of course, these evidences of cerebrovascular impairment bring from the patient urgent requests for treatment and for "doing something" about his disease. However, we do not regard them, especially with adequate treatment, as indicating a hopeless outcome.

Likewise, severe retinopathy, particularly papilledema, may give a clue that the patient has so-called accelerated or malignant hypertension. This may make the physician feel that he must do something, since if it is untreated, this disease usually runs a course less than two years from its onset. Nevertheless, retinopathy may be the most striking and the earliest of all the signs to improve on successful treatment. Certainly it does not argue against a good result with treatment.

Patients who are over 50 years old, particularly if they have

high diastolic pressures (we pay little attention to the systolic pressures in the elderly), have an unfavorable outlook as compared with younger patients with the same pressures. Women tolerate the disease better than men. Obesity, particularly if the patient will not reduce, may be unfavorable; but if the patient will reduce, this may be a favorable sign.

Some prognostic significance is given to the diastolic pressure, particularly when it is over 140, 150 or 160 mm. Hg, although this is not a terribly helpful sign. We are more inclined to pay attention to the blood pressure if it is "fixed", that is, if it will not decrease on bed rest or sedation. The sign of a rapid pulse rate is favorable and this is more common among women. In other words, other things being equal in the untreated patient, a slow pulse rate is less desirable from the prognostic point of view.

What is the treatment for hypertension? The medical staff of our clinic is particularly interested in medical treatment which, of course, involves the use of drugs. However, one associate, Dr. Smithwick, is the leading surgeon of the world in the treatment of hypertensive disease by sympathectomy. Hence, many cases are observed with him and we know a good deal about that type of therapy.

The therapeutic schedule employed is the so-called "up-the-steps, down-the-steps" regimen, and the patients are placed on this when they have relatively "benign" or gradual hypertension that will not moderate on reassurance. This treatment is more appropriate for the patient if he shows signs of progression. Obviously, this means that the patient must be seen on a number of occasions.

The first drug of choice, because it is the mildest, is Rauwolfia or one of its derivatives, such as reserpine. There is no great advantage in using the crude drug or one of the derivatives. Secondly, if Rauwolfia alone is unsuccessful, as it may be, another hypotensive drug is added but not substituted for it. The procedures employed are nonspecific hypotensive ones and work well additively. If the pulse is still rapid after Rauwolfia, which tends to slow the pulse rate, veratrum is given which also tends to slow the pulse. If, however, the pulse is slow the second step is usually omitted and the course proceeds to the third step employing hydralazine. This drug increases the pulse rate and has other

side effects which can be obviated by instituting it gradually. It is advisable to avoid the pulse-rate-accelerating effect, if possible, through the prior use of Rauwolfia and, if necessary, veratrum.

These three drugs together, in gradually increasing dosages, frequently are quite effective when continued over a period of weeks or months. One should be prepared to continue the treatment in this disease for long periods and not expect a miraculous response overnight. This type of approach has convinced us of its effectiveness.

The use of ganglionic blocking agents is usually postponed for use at the end of our regimen because of their troublesome characteristics. If it were necessary to choose one blocking agent, and there are many of them, we would begin with pentolinium, because in our experience it is better tolerated on the average than any of the others. However, it is not strikingly superior and the preference for it is not great. If it is not well tolerated, another blocking drug is substituted without much delay.

As an example of the method, in one patient, a young woman with a very bad family history, all treatment was omitted and she did well for some years. Then she began to have symptoms, particularly headaches, and treatment was started with crude Rauwolfia in the form of Raudixin® which was later replaced by reserpine to determine whether that derivative was equally good. Its effect seemed to be the same, but with a very small dose of hydralazine added — only 25 mg. four times a day, or a total dose of 100 mg. — the blood pressure became perfectly normal. The effectiveness of this regimen was proven by the temporary substitution of placebos. She is now continuing, three years later, on this schedule, working everyday; and although it cannot be proved that her life has been prolonged, it is at least a better one.

When hexamethonium is introduced into the picture, there is a well demonstrated necessity for close observation of the postural effects of a blocking agent such as this. The pressure is usually somewhat lower in the standing position. In one case, the patient had severe hypertension with blood pressure ranges of 240/140 to 240/150 mm. Hg. Using reserpine and hexamethonium, there

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was a very striking lowering of the blood pressure and following a hot bath the patient had postural syncope with a severe complicating head injury. Reactions of this type are one of the reasons why these drugs should be discontinued, if possible.

After the above unfortunate episode, hydralazine in the dosage of 100 mg. four times a day was employed with the same dosage of reserpine. There was a good response in maintenance of a satisfactory blood pressure level and the therapy was interrupted to be sure this was a therapeutic response rather than a spontaneous remission. This was repeated twice in order to prove that these drugs were indeed very potent hypotensive agents in this patient.

One of the features of this therapeutic method to be emphasized is the progressive change in drugs called "coming down the steps". Patients are first tried on the step series of additive combinations of antihypertensive drugs, adding one drug to the other, only if and as necessary, to achieve the desired lowering of the blood pressure. Once that is done, the desirable pressure is maintained, if possible, for some weeks and preferably months, after which, the elimination of drugs is attempted. It is particularly desirable to discontinue the blocking agents first and then to reduce the dose of hydralazine because this is a potentially toxic drug.

It is remarkable to see how many of the patients can be brought down to minimal doses of Rauwolfia or reserpine alone and held there. In simple language, this could be stated as follows: "It is easier to keep the pressure down than it is to get it down." Naturally, it is necessary to keep it down, but the administration of Rauwolfia or reserpine is interrupted to see if it will become elevated again. In our experience, it always will in familial hypertensive disease. Therefore, there is apparently no particular loss to the physician or to the patient in continuing small doses of Rauwolfia which can be maintained in many persons for years without symptoms. However, one must be prepared to reinstitute or increase doses of other drugs if the pressure begins to increase. It is particularly important to remain alert to adverse changes in the blood pressure level when other hypertensive factors may enter the picture, for example, cold weather or emotional stress. This is true in the autumn season when the doses are increased

or the use of a drug is reinstituted. Contrariwise, there is an obvious advantage in beginning the treatment of hypertensive patients in the spring season, and as summer approaches the vasodilating effect of warm weather seems to have a synergistic action with the drugs. As mentioned, nervous, emotional, and other stimuli likewise must be taken into account during this regimen.

What can be achieved under this type of regimen? The results were collected on 137 cases that did not respond to Rauwolfia alone. These could be called resistant cases under the criterion that Rauwolfia alone is a fairly effective hypotensive agent. In general, the therapeutic procedure in each case followed the steps of Rauwolfia plus veratrum, Rauwolfia plus hydralazine, then the combination of the three, and finally, if necessary, those three with the addition of hexamethonium. The more severe the degree of hypertension, judged on the basis of average blood pressures, the more extensive is the treatment usually required. One striking feature in this group is that a third of them, or 45 of the 137 patients, were able to continue on Rauwolfia alone after discontinuation of the combination of drugs. Furthermore, the results in those patients were satisfactory in that the average blood pressures were within the range of 150/90 mm. Hg or 160/90 mm. Hg, considered to be normal. This was nearly as good as the results in the other more severe groups.

The group of patients with the highest blood pressure averages did not do as well and these continued to require hexamethonium which, of course, was anticipated. The step by step elimination of drugs, therefore, seems to be an effective method of reducing therapy which may not actually be needed in hypertensive disease.

Two new drugs should be mentioned, one of which has recently had wide publicity in the daily press. The first is the drug called BAS, the benzyl analogue of serotonin. This substance, serotonin, appears to have some definite relationship to the action of reserpine and Rauwolfia so that simpler analogues of these materials were made and one of them is this benzyl analogue of serotonin which is abbreviated into BAS.

This drug seems to be a simple form of reserpine, pharma-

cologically as well as chemically. Its clinical effect is the same as that of reserpine with one or two differences. This was exemplified in one very unresponsive patient who was started on reserpine, after which were added ample doses of hydralazine and veratrum; but the combination produced little response, the blood pressure continuing between 180 and 200 mm. Hg systolic and about 100 to 110 mm. Hg diastolic. When BAS was added, it responded and came down to about 150/90 mm. Hg or 160/90 mm. Hg, and the patient felt a great deal better.

This drug is interesting, therefore, in that it may reveal the mechanism of action for reserpine. It would be well to remember the word "serotonin" because more information may become available on it. It exists in the brain and elsewhere as probably a very important hormonal or humoral substance regulating possibly the blood pressure and other bodily functions. BAS is, therefore, another hypotensive drug.

Another new drug was effective in reducing the blood pressure of the man mentioned above completely to normal, 120/80 mm. Hg. It has been known for some time that the kidney has something to do with the blood pressure. As already indicated, there are clearly certain forms of renal hypertension and the classic experiment of Goldblatt is well known. By interfering with the circulation through the kidney, it is possible to produce in animals hypertension of long duration and severe degree that otherwise simulates almost exactly what we call essential hypertension in man. This explains our concentration on the question of what rôle the kidney may play in essential hypertension. In so-called malignant and other severe forms of hypertension, the onset of even incipient congestive heart failure made these patients very resistant to therapy. Therefore, one of my associates, Dr. Hollander, and I have been working for a number of years with the adjunctive use of diuretics in the treatment of hypertension in patients of this type. Some of them did become quite sensitive to therapy after the strong diuretic effect of mercury administered intravenously.

Intravenous mercury, as is well known, is difficult and perhaps dangerous to use repeatedly. Some observers feel that its effects are essentially toxic. At any rate, we preferred the use of an oral

diuretic and have tried every one that has been produced over the past four or five years. One of these is called chlorothiazide, an oral diuretic which has no mercury in it. It is a diazine derivative containing two benzyl rings and was produced first as a carbonic anhydrase inhibitor. However, it is understood from those who are proficient in this field that this compound is a relatively weak carbonic anhydrase inhibitor, and while it acts as a diuretic its mode of action is not completely understood. Incidentally, it is a very effective oral diuretic. In our experience, it is as effective as parenteral mercury and, indeed, may act when the patient becomes mercury-fast. This refers to patients having congestive heart failure with edema.

As our use of this material increased, it became apparent that its action was also hypotensive, but most strikingly so when used to sensitize a patient to the action of the standard hypotensive drugs. In one patient with severe hypertension of 250/150 mm. Hg, and whose response was very disappointing on the step regimen, the pressure definitely came down with the addition of chlorothiazide, although the dosage used was slight. In another patient, we were using hydralazine alone because there was no striking evidence of a pentolinium effect. At this point, chlorothiazide was added with a very striking reduction in blood pressure, practically to the normal level. Then, hydralazine was discontinued with only a slight rise in the blood pressure while on chlorothiazide alone. Placebos were substituted to provide conclusive evidence that this was not a spontaneous response. However, on resuming the dosage of chlorothiazide alone, it was obvious that the blood pressure level was definitely affected by this drug. In many cases like that, the drug seems to be hypotensive alone but also to be most effective when given in combination with other hypotensive treatment. It is concluded, therefore, that chlorothiazide is a more potent hypotensive agent than any of the other oral diuretics. Another orally effective diuretic agent, which is a triazine, was received for study. It is considered to be good in congestive heart failure, but in our experience it has not been proved to be hypotensive. If it has this effect, it is a very weak one. Therefore, chlorothiazide has unique hypotensive properties or stronger hypotensive action than the other available diuretics.

It was further observed that during the oral administration of chlorothiazide over a period of weeks and months, it ceases to be primarily a diuretic, symptomatically at least. It is usually a diuretic early in the course of its administration, but this action is soon lost. Any weight lost by patients is then regained. They are not kept up at night with nocturia, nor do they have polyuria during the day; so, at least from their point of view, the drug is free of diuretic symptoms. Indeed, used in small dosages, which are about one-half those necessary or usually employed for its most potent diuretic action, the drug is virtually asymptomatic. It produces few or no side effects. This statement is a carefully considered one because one must always leave the loophole that it may produce symptoms. The symptoms observed while using it for ten months have seemed to us to be associated more with the hypotension that ensues, particularly from blocking drugs, than from the drug itself. Therefore, we believe that this drug is a most interesting adjunct to our hypotensive armamentarium.

There is likely some additional action on the kidney not well understood at present. There is evidence that when it is used with other drugs it acts against factors blocking the response that might otherwise occur from use of the other antihypertensive drugs.

Its action following bilateral lumbodorsal splanchnicectomy has been surprisingly satisfactory in lowering the blood pressure. One patient with malignant hypertension, papilledema, headache, and impairment of the intellect was placed on our usual regimen for this type of case. This began with the immediate use of a blocking drug which increased the blurring of vision and produced dryness of the mouth, generalized weakness, and very marked orthostatic hypotension, along with other unpleasant side effects. These occurred in spite of the substitution of chlorisondamine for pentolinium, reserpine being continued. As a result, the patient insisted that the regimen be discontinued and the bilateral splanchnicectomy was done. This did not produce the desired lowering of blood pressure and the administration of chlorothiazide was started as we had just learned of its strong adjunctive properties. The concept that it might be strongly adjunctive to surgery was verified and we can say now that it is more striking in patients with sympathectomies than in any other group. The

patient is now being maintained with normal blood pressure by the use of relatively small doses of the drug. It should be noted that it does not seem to have a deleterious effect but rather it may, perhaps, have a beneficial effect on renal function.

It is too early to say for certain what this type of approach to the therapy of hypertension will do, from *your* point of view, which is the long term outlook for hypertensive individuals. These impressions reported here are from our clinical experience. Not only do these patients have lower blood pressure but they are markedly improved subjectively. They express themselves freely about their degree of improvement and give the impression that some sort of pressor mechanism has been interrupted, particularly when chlorothiazide is added to other hypotensive procedures.

It does not appear that chlorothiazide alone is as potent perhaps as the blocking agents. However, it can be said that chlorothiazide in conjunction with other treatment, is as potent if not more so than the blocking drugs and has few or none of their side effects.

PRESIDENT GETMAN — Thank you very much, Dr. Wilkins. I am sure we all found that most interesting.

I have asked Dr. Arthur Parks to discuss Dr. Wilkins' presentation.

DR. ARTHUR E. PARKS — I am sure you agree with me that it has been a great privilege to have heard Dr. Wilkins bring us up to date on an extremely difficult and involved subject, one that is beset with contradictions.

We, as medical directors, are interested in long term expectancy of life, and I believe it is true to say that life expectancy in the hypertensive patient may be said to be inversely proportional to the level of the blood pressure. That is a general statement, and Pickering has proved its validity. However, we know, too, that with hypertension it is extremely difficult to generalize because there are so many exceptions and there are so many people who have essential hypertension, who seem to carry it well without symptoms and live to a normal life expectancy.

We all are interested in the cause of death in hypertension. Well, we know what that is. It is thrombosis and it is hemorrhage, be it

in the heart or the brain or the kidney. We have to ask ourselves what causes that. I think the answer was shown many years ago by Professor Turnbull of London that it is arterial degeneration. What causes arterial degeneration? There, again, we do not know, so we are left with questions that are unanswered.

Attention, then, has been shifted to more remote causes, and I believe that heredity comes into the picture at this point. We are greatly indebted to Professor Pickering of Oxford and to Dr. Caroline B. Thomas of Johns Hopkins for their recent researches into the genetic factor in hypertension. And so, we have a worldwide picture of people everywhere attempting to solve this very difficult problem.

Dr. Wilkins has been one of the leaders in this field, and he has attacked the immediate problem of dealing with patients who have elevated blood pressures. He is attacking it in the belief that if we lower the blood pressure, we will help the patient and prolong his life expectancy. Here, one sees difficulties.

If we have a patient with full-blown malignant hypertension to deal with, he has high blood pressure with symptoms, and Dr. Wilkins has indicated to us that he will attack such patients quickly with ganglionic blocking agents. More difficult is the patient who has high blood pressure, perhaps 220/120 mm. Hg, but who has no symptoms, and where all one can find is mild cardiac hypertrophy. Sometimes the aorta seems to give way; at other times it is the left ventricle that gives way. All of these, as he has shown us, are carefully assessed when he first sees the patient.

Well, no one can deny that great strides are being made. He has shown us many examples of how patients feel better and are better when their blood pressure is lowered. We, as medical directors, have to say, "Yes, that is true, but the numbers are few." He himself has just told us that statistical evidence on large numbers of cases is not yet available. We will look to him and to men like him for that statistical evidence that life is, indeed, prolonged in large groups. When we have that knowledge, and only then, are we in a position to say that arterial degeneration which causes death is, indeed, delayed and that, perhaps, in the future we may be able to deal more leniently from an underwriting point of view with patients who have hypertensive disease.

Dr. Wilkins, it affords me very much pleasure to thank you for your address, and I sincerely hope that we may hear from you again in the future.

PRESIDENT GETMAN — Thank you very much. Are there any questions that anyone would like to ask?

DR. BARTHOLOMEW A. RUGGIERI — Just as a matter of curiosity, Dr. Wilkins, what did you mean by the adrenogenital syndrome? Did you mean macrogenitosomia praecox or pseudohermaphroditism?

DR. WILKINS — Well, I hope it will not surprise you when I tell you that I do not know what I mean. We do see such patients, particularly among children, and this is entirely out of my field. I must say that I never see pediatric patients, but my friends tell me that there are children who have what they refer to as "adrenogenital syndrome". There are young women, the hairy young women, who sometimes have hypertension as a portion of their syndrome. That was included merely to indicate that we do believe other adrenal disturbances exist besides the ones mentioned, namely, pure Cushing's disease, pheochromocytoma or primary aldosteronism, which seem to have hypertension as a part of the picture.

DR. RUGGIERI — The reason why I ask that is that the two conditions I mentioned are due to a congenital hyperplasia of the adrenal cortex.

DR. WILKINS — Yes, that is true; and, again, I am not familiar with this syndrome in youngsters. As you know, some of the cases of Cushing's disease are treated by subtotal adrenalectomy, where they also have hyperplasia of the adrenal cortex.

DR. RUGGIERI — I am a pediatrician and part time worker in insurance, and I know, Dr. Wilkins, the cases that I have seen clinically usually have an increase in sex hormone disturbance, associated or not associated with a decrease in the salt and water hormone disturbance. I was curious because I have certainly not seen hypertension in these cases. With the possible decrease in salt and water hormone disturbance, one would not expect hypertension, at least not on theoretical grounds.

DR. WILKINS — I have no comment.

DR. WALTER C. HAUSHEER — I would like to ask Dr. Wilkins whether he has any technic that could be generally applied to the urine examination to determine whether or not a patient is taking any one of these drugs, and which would be applicable in large numbers of specimens?

DR. WILKINS — This is a very practical question and one that is asked by those who are disturbed about patients who may take these drugs and pass examinations without the knowledge of the examining physician.

I do not know. Let us begin there because it is a factor in which we have not interested ourselves. Perhaps you people should develop methods for identifying the patients or the people who might be taking these drugs.

In the absence of such chemical methods, there are patients in whom it is not possible to tell that they ever had hypertension. Many of these have been severe hypertensives, and they are still on drugs. I think you have a problem there, but how you are going to deal with it I do not know.

DR. MURRAY F. BELL — Dr. Wilkins, in the early part of your talk, I believe, you mentioned that a slower pulse is a more unfavorable sign than a more rapid one, which you said you would comment on later.

DR. WILKINS — Let me tell you a favorable type of patient, regardless of blood pressure. She is a young woman with two children, who is a Cub Scout leader and a typical matron with no help, and has a family history of hypertension. She comes in to you with a red face, a fast pulse, and a high blood pressure.

I always feel quite confident in my mind that we are going to get a good result with that type of patient both in lowering the blood pressure and symptomatically. In other words, clinically they are easy cases to treat, and I think that part of the success must involve the rapid pulse rate.

The reason for this is unknown but I would rather see that type of patient than the same patient with the identical blood

pressure but a pulse rate in the 60's. In our experience, slowing the pulse rate, particularly with Rauwolfia, is nearly always accompanied by reduction in the blood pressure.

We like to reduce the pulse rate, particularly if we are going to use hydralazine, which may affect the heart causing palpitation and tachycardia. Thus, unless there is congestive failure, a rapid pulse rate in a new patient is, in general, evidence that a good result with therapy can be expected.

PRESIDENT GETMAN — Our next two papers, I hope, will be of great interest to all of us. They will not be discussed, but questions will be invited on both of them; and if you have any questions, I wish you would withhold them until the second paper has been given.

Our next paper is a presentation of a study by our colleagues at the Mutual Life Insurance Company of New York, Dr. Albert A. Pollack, Dr. Thomas J. McGurl, Jr., and Dr. Theodore E. Plucinski. It is entitled, "Hypertension in Substandard Insurance". This will be a most valuable contribution to our program. Dr. Pollack will present the paper.

HYPERTENSION IN SUBSTANDARD INSURANCE*

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In 1907 Dr. Theodore C. Janeway[1] published a monograph, "The Clinical Study of Blood Pressure", which was probably the earliest American report on a blood pressure study. In this he practically ignored diastolic pressure while devoting most of the book to the systolic phase. In 1940 Hines[2] of the Mayo Clinic reported results in a follow-up survey of 1,522 patients twenty years after the initial visit. He found that 85 mm. Hg diastolic pressure marked the critical level and that a higher diastolic pressure was frequently followed by the development of clinical hypertensive disease in later years.

The difficulty in evaluating blood pressure readings is well known. A poorly placed cuff can vary the reading as can a leak in the air or mercury system. The subject should be relaxed, but this is not easily accomplished when he is being examined for life insurance, and it is a rare insurance applicant who is not tense. Hines[3] found that blood pressure is unequal in the two arms in about 50 per cent of normal individuals. It averaged about 13 mm. Hg in the systolic and diastolic differences. These changes were transient or variable and were not present in the same degree on successive observations. This was illustrated in a series of persons with essential hypertension whose resting blood pressure averaged approximately 50 mm. Hg lower on subsequent visits than when first observed.

Master and his co-workers[4] published "Normal Blood Pressure and Hypertension" in 1952. Their data were collected on a

*Grateful acknowledgment is made to Dr. J. R. Gudger, Mr. N. Macintyre, Mr. W. Schmidt of The Mutual Life Insurance Company of New York and Mr. R. J. Johansen of Metropolitan Life for their cooperation and help in preparing this manuscript.

segment of industrial employees and they give a normal range of systolic and diastolic pressure for each age and sex as well as the lower limits of pressure which they regard as representing true hypertension for these groups. They concluded that blood pressure normally rises with age in both sexes and that our current concept of hypertension is often incorrect or misleading because of the low dividing point now accepted.

Much of our present day information about arterial blood pressure is based upon various investigations made by life insurance companies. The present study was intended to evaluate our company's practice in underwriting blood pressure impairments. Our aims were to determine:

1. Whether lability was a significant factor in the mortality from hypertension.
2. Whether our method of averaging blood pressures (using a prior high reading and current examination readings) is a correct underwriting procedure.
3. The causes of death among the individuals in a group known to have had hypertension.
4. The significance of age on mortality in this group of policyholders with abnormal blood pressure.

The material used in this investigation was taken from files on policyholders who were substandard risks (requiring increased premium rates) solely because of abnormally elevated blood pressure. In all cases, at least one reading was above 136 mm. Hg systolic or 88 mm. Hg diastolic. The analysis, therefore, included only those cases with a blood pressure average high enough to require an extra charge in premium, based on the currently estimated additional risk assumed. Further, all individuals with any other impairment requiring an extra premium were excluded. This means that conditions like obesity or other metabolic disorders, demonstrable cardiovascular changes, and diseases of the nervous, digestive and renal systems — or a history of these — were not considered to be severe enough to affect longevity if they existed in any case. No women were included because of the small number. The final series was composed of information on 2,510 policies issued between 1942 and 1954.

The importance of the manner by which the final blood pressure figures were obtained is obvious. The procedure followed is described in the following summary.

If the first current reading was above the average (requiring a mortality debit), according to tables based on combined company experience, a second reading was obtained and the average of these used.

If the variation between the two current readings was over 20 mm. Hg systolic or 10 mm. Hg diastolic a third was obtained, the average of the three being used.

A prior reading (highest systolic and highest diastolic from any authentic source) was averaged with the required current readings if the average was thereby increased.

The examining physician took all blood pressures with the subjects seated and the final average, as explained above, was used in the risk evaluation.

In our series of cases the highest and lowest systolic, and highest and lowest diastolic, recorded within a period of six months, was accepted as the range of the blood pressure for that person.

Lability of Blood Pressure

An analysis of the material to determine the accuracy of our underwriting procedure, as well as the significance of lability in the mortality of hypertension, is shown in tables 1, 2, 3 and 4. It is apparent that any bizarre variation in multiple readings would introduce a certain amount of distortion in the final analysis of the material. This effect was avoided by deliberately choosing a homogeneous group of cases in which the average of the high and low *diastolic* pressure readings were within 5 mm. Hg of the average diastolic pressure used to classify the life insurance risk. These were then subdivided into two groups having either labile or nonlabile blood pressure characteristics.

The first group included those with a range of 12 mm. Hg or less in the diastolic pressure (*nonlabile*), and the second group those in which the range was greater than 12 mm. Hg (*labile*). These were then listed according to the average pressures within the groups as shown in table 1. The differences in mortality

TABLE 1

Comparison of Mortality Experience with Labile and Nonlabile Diastolic Blood Pressure Elevations

Average Diastolic Pressure mm. Hg	Expected Deaths at Substandard Class*	Actual Deaths (Policies)	Per Cent of Actual to Expected Deaths
Group I (0-12 mm. Hg Range)			
80	4.93	10	203
86	11.13	9	81
90	16.60	48	289
96	11.02	23	209
100	3.59	9	251
106	1.29	3	233
110	0.08	0	0
Total	48.64	102	210
Group II (13 or more mm. Hg Range)			
80	0.94	2	213
89	0.99	1	101
92	3.85	5	130
95	1.47	2	134
102	0.86	5	581
107	0.02	0	0
Total	8.13	15	185

*From Intercompany 1946-1949 Select Mortality Table.

experienced between group I (nonlabile blood pressures) and group II (labile blood pressures) are not statistically significant. Table 2 illustrates the same division of cases for the *systolic* blood pressures. Here, 15 mm. Hg was used as the separation point between labile and nonlabile groups. Again, no statistical difference was found between the two groups.

Tables 3 and 4 illustrate the results of several groupings as described above, in an effort to determine whether there was any difference in mortality ratios at a specific range between high and low systolic and diastolic blood pressures. As can be seen, all the mortality ratios are close and there is no statistical significance in the differences.

It is fairly obvious in this series that the mortality in the

TABLE 2

Comparison of Mortality Experience with Labile and Nonlabile Systolic Blood Pressure Elevations

Average Systolic Pressure mm. Hg	Expected Deaths at Substandard Class*	Actual Deaths (Policies)	Per Cent of Actual to Expected Deaths
Group I (0-15 mm. Hg Range)			
130	3.82	7	183
140	21.05	52	247
150	14.24	23	162
160	4.57	10	219
170	0.33	1	303
180	0.07	0	0
Total	44.08	93	211
Group II (16 or more mm. Hg Range)			
130	0.66	1	152
140	5.20	13	250
150	5.15	13	252
160	0.74	0	0
170	0.05	1	2000
180	0	0	0
Total	11.80	28	237

*From Intercompany 1946-1949 Select Mortality Table.

TABLE 3

Comparison of Mortality According to Increase in Variation of Diastolic Pressures

Groups	Variation of Pressure Range in mm. Hg	Expected Deaths at Substandard Class*	Actual Deaths (Policies)	Per Cent of Actual to Expected Deaths
I	0–5	30.09	58	193
II	6 or more	26.68	59	221
I	0–8	40.59	81	200
II	9 or more	16.18	36	222
I	0–12	48.64	102	210
II	13 or more	8.13	15	185

*From Intercompany 1946-1949 Select Mortality Table.

TABLE 4

Comparison of Mortality According to Increase in Variation of Systolic Pressures

Groups	Variation of Pressure Range in mm. Hg	Expected Deaths at Substandard Class*	Actual Deaths (Policies)	Per Cent of Actual to Expected Deaths
I	0–5	21.89	45	206
II	6 or more	33.99	76	224
I	0–10	38.58	75	194
II	11 or more	17.30	46	266
I	0–12	40.58	82	202
II	13 or more	15.30	39	255
I	0–15	44.08	93	211
II	16 or more	11.80	28	237

*From Intercompany 1946-1949 Select Mortality Table.

groups with nonlabile blood pressure differed very little from the ones with labile blood pressure. It is also evident that our method of averaging the blood pressures is the correct means of arriving at an underwriting appraisal of the risk involved.

Life Expectancy

The life expectancy for individuals with various high diastolic and high systolic blood pressures is shown in tables 5 and 6. The over-all ratio of actual to expected deaths in the Intercompany 1946-1949 Select Mortality Table was computed for each group. Mortality tables were constructed for that percentage mortality and the expectations of life were computed from these, using the standard technics. For convenience in these computations age 40 was taken as representative of the 30-49 age group, and age 55 for the group 50 and over.

TABLE 5

Life Expectancy Based on 1946-1949 Select Mortalities

Diastolic Pressure

Classification	Per Cent Actual to Expected Deaths		Life Expectancy (Years)	
	Age at Issue		Age at Issue	
	40	55	40	55
Unimpaired risks	100	100	33.1	21.5
Under 89 mm. Hg	180	266	27.7	14.1
90 mm. Hg and over	391	234	20.9	15.5

TABLE 6

Life Expectancy Based on 1946-1949 Select Mortalities

Systolic Pressure

Classification	Per Cent Actual to Expected Deaths		Life Expectancy (Years)	
	Age at Issue 40	55	Age at Issue 40	55
Unimpaired risks	100	100	33.1	21.5
Under 150 mm. Hg	288	155	23.0	18.3
150–159 mm. Hg	362	254	22.0	14.7
160 mm. Hg and over	460	307	19.9	13.5

For one fourth of the cases, issue age under 30 years, there were too few deaths to be statistically significant and hence the ratios were not computed.

The expectancy of life is shorter by 6.8 years (table 5) in the age 40 group when *diastolic* pressure was 90 mm. Hg or over, as compared to lower levels. However, no such effect was evident in the older group, age 50 and over. *Systolic* elevations in these age groups were identified with a gradual slight decrease in life expectancy as the pressure increased. Table 6 illustrates these findings.

Causes of Death

From tables 7 and 8 it is apparent that the largest number of cases, about half the total, fell in the 30-49 year age group. The number of cases under 30 years and over 49 years was about evenly divided, each representing roughly one quarter of the series. This age distribution is similar to that among policyholders who are accepted as unimpaired risks paying standard premium rates. There were 120 deaths among the 2,510 policyholders with a blood

TABLE 7

Age Distribution and Number of Deaths by Diastolic Blood Pressures

Age in Years	Lives		Deaths	
	under 89 mm. Hg	90+	under 89 mm. Hg	90+
under 30	206	233	0	4
30–49	341	1241	7	60
50 and over	102	387	14	35

TABLE 8

Age Distribution and Number of Deaths
by Systolic Blood Pressures

Age in Years	Lives			Deaths		
	under 150	150–159	160+	under 150	150–159	160+
under 30	164	161	114	1	3	0
30–49	715	484	383	27	20	20
50 and over	117	186	186	9	21	19

pressure impairment. Thus, 4.8 per cent of these persons died during the period from 1942 to 1954. Despite the fact that a considerable number of cases was studied none had severe degrees of hypertension, since these were excluded by the process of life insurance risk selection. Table 9 illustrates the age distribution of these deaths. At the time of death, 3.6 per cent were under 30 years of age; 55.6 per cent were between 30 and 49 years old; and 40.8 per cent were over 50. Correcting for exposure, these figures follow the expected trend in the three broad age groups, the number of deaths progressively increasing with age.

TABLE 9

Deaths in Age Groups

Age in Years	Number of Deaths	Percentage
under 30	4	3.6
30–49	67	55.6
50 and over	49	40.8
2510 cases — 120 deaths in this group		4.78%

In table 10 the causes of death are listed and in some the percentage of relative frequency was compared with the same cause of death in a group of society more closely approximating the general population. These are the Industrial Premium-Paying policyholders of the Metropolitan Life Insurance Company of New York. The incidence of coronary artery disease as the cause of death was slightly higher in our study as it was also for malignant hypertension; but it is interesting to note that the percentage for cerebrovascular lesions was approximately the same. Suicide was a less frequent cause of death in our study but malignancy was equal in both groups.

TABLE 10

Causes of Death	Number of Deaths This Study	Percentage of Total Deaths Metropolitan Life Insurance Co.*	Percentage of Total Deaths This Study
Accidental Death	5		4.2
Cerebrovascular Lesions	13	10	10.9
Coronary Artery Disease	69	51	57.5
Malignancy	11	9	9.0
Malignant Hypertension	4	2	3.3
Suicide	3	4	2.6
Other Causes (not related to cardiovascular disease)	15		12.5
Total:	120		100.0

*Deaths in white males, ages 20 years and over during the year 1954, from the Industrial Premium-Paying policies of the Metropolitan Life Insurance Company of New York.

Conclusion

Before drawing any conclusions from a study such as this, it is important to emphasize that these cases composed a group of highly selected risks. We were concerned only with elevations of blood pressure — that is, *mild* essential hypertension. Any other significant impairment at the time of issue was cause for excluding the case from the study. Those with *moderate* and *severe* degrees of hypertension were also excluded in the process of life insurance risk selection.

There was no evidence that lability was a significant factor in the mortality from hypertension. The mortality ratios were about the same for the labile as for the nonlabile groups in which the systolic and diastolic pressures were analyzed separately. Thus, the average degree of elevation was as significant as the range of variation in blood pressure. An occasional elevated reading should be included in the determination of the average blood pressure for an individual.

There were no startling conclusions to be drawn from the analysis of the causes of death. It is our opinion that the differences between percentages in this study and those of the Metropolitan

Life series, which more nearly represents the normal male population, were relatively greater than the actual differences.

The significance of mild hypertension in the selection of life insurance risks seems to diminish somewhat as the age increases among the groups in this investigation.

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PRESIDENT GETMAN — The next paper is entitled, "Mortality Study in Hypertension", and is by Dr. William Bolt, Chief Medical Director, and Dr. Murray F. Bell, Associate Medical Director, of the New York Life Insurance Company, in collaboration with Dr. Jack R. Harnes, Clinical Instructor in Medicine at New York University College of Medicine.

A STUDY OF MORTALITY IN MODERATE AND SEVERE HYPERTENSION*

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This report concerns the mortality experience on a group of 3,283 life insurance applicants with a sufficient degree of arterial hypertension to cause their rejection or their acceptance with substantial premium increases. The investigation was designed to bring out information regarding the prognosis of people with hypertension that might be helpful both in insurance and clinical medicine.

Method

The material consisted of white applicants who, when examined for life insurance during the period from January 1, 1946 through December 31, 1950, had a blood pressure exceeding 162 mm. Hg systolic and/or 102 mm. Hg fifth phase diastolic. The selection of cases for study included those with enlargement of the heart, abnormal electrocardiogram, excessive weight, or tachycardia. However, those with any other impairment, even minor ones of questionable significance, were excluded.

Each of the 3,283 cases thus selected for analysis was followed through 1956. Pertinent information was abstracted from the files, including all blood pressures and pulse rates available from our company records, as well as those of other companies. Fourth phase diastolic pressures recorded prior and subsequent to entrance into the study were converted to fifth phase diastolic

* We wish to acknowledge the valuable assistance and advice of the Actuarial Research Division of New York Life Insurance Company and in particular that of Mr. Joseph Sibigtroth, Mr. Donald Hillenmayer and Miss Nora Beattie.

pressures by subtracting six points. The date of onset of hypertension was obtained from insurance records, applicants' statements, or replies from private physicians.

The dates and results of any electrocardiograms in the files were recorded. These were classified as normal (including minor variations of questionable significance) or abnormal. Transverse cardiac diameters computed on teleoroentgenograms to be over +12 per cent by the Ungerleider-Clark scale were considered to be enlarged.

Previous abnormal electrocardiograms and chest x-rays were accepted as evidence of abnormality at the time of the examination. Subsequent normal ones were considered presumptive evidence of normalcy at the time of the examination. Subsequent abnormal electrocardiograms and chest x-rays were discarded since their status at the time of the examination could not be determined.

The blood pressure records were analyzed by the following methods of grouping:

1. Highest systolic pressure on current examination. Highest diastolic pressure on current examination.
   (Any repeat observations were considered part of the current application and included in the current blood pressure.)
2. Average of all systolic blood pressures during the past five years. Average of all diastolic blood pressures during the past five years.
   (In this second group, and in subsequent categories, the average of all blood pressures on a single day was used as a single observation.)
3. Average of the two highest systolic blood pressures during the past five years. Average of the two highest diastolic blood pressures during the past five years.
4. Average of all current systolic blood pressures. Average of all current diastolic blood pressures.
5. A mean blood pressure (the diastolic pressure plus one third of the pulse pressure) was calculated for the average of current readings and for the highest and lowest current readings.

As a measure of blood pressure lability, a record was made of any case in which the systolic pressure fell below 140 mm. Hg and the diastolic below 90 mm. Hg. A contrasting group in which subsequent pressures did not fall to normal was also noted.

Information as to whether the applicant was living or dead was obtained from various insurance records, contact with the individual, his family or acquaintances by letter or telephone, and information from attending physicians. In a few instances more extensive efforts were required. Only 48 or 1.5 per cent of the 3,283 persons could not be fully traced. Of these, some information was available in 25 cases and these were treated as lapsed cases and included in exposures of the various groups. It is not felt that the absence of complete information about these 48 individuals will significantly affect the results.*

In analyzing the results, expected deaths were based on the standard life insurance experience of a group of large companies during recent years of issue.

Results and Discussion

Table A shows a summary of the entrants of whom 2,497 were men and 786 women. Among these, 414 men and 79 women were known to have died before the end of 1956. It will be seen that the untraceable and partially traced individuals are reasonably well distributed in the various age groups.

The over-all mortality ratio (actual to expected deaths) was 270 per cent.

Factors in Evaluating Blood Pressure Readings

The mortality ratios resulting from the various methods of utilizing blood pressures are shown in tables 1(a) through 4(c) and charts 1(a) and 1(b). These indicate that the methods utilizing the average of current systolic and diastolic readings, the average of all blood pressures during the past five years, the average of the two highest blood pressures in the past five years and the highest systolic and diastolic blood pressures on examina-

* This degree of success in tracing 98.5 per cent of individuals who for the most part were declined or had refused the rated policies, was due to the untiring efforts of Miss Winifred Young, whose diligent search for these individuals made this study possible.

TABLE A

SUMMARY OF ENTRANTS

Entry Age	Living	Deceased	Traced in Part	Completely Untraced	Total
		Men			
39 & less	656	60	4	2	722
40–49	682	126	7	6	821
50–59	579	166	2	6	753
60 & over	138	62	1	0	201
Total	2055	414	14	14	2497
		Women			
39 & less	47	1	1	1	50
40–49	250	17	4	1	272
50–59	314	39	6	6	365
60 & over	76	22	0	1	99
Total	687	79	11	9	786
		Men and Women			
39 & less	703	61	5	3	772
40–49	932	143	11	7	1093
50–59	893	205	8	12	1118
60 & over	214	84	1	1	300
Total	2742	493	25	23	3283

TABLE 1(a)
AVERAGE OF CURRENT SYSTOLIC AND DIASTOLIC READINGS
Mortality Ratios (Actual Deaths in Parenthesis)
Men

Age	Systolic	Diastolic 5th Phase Reading: 97 & less	98–107	108–117	118 & over	Total
39 & less	157 & less	396% (7)	396% (6)	1358% (5)	—% (0)	487% (18)
	158–177	176 (4)	658 (9)	1099 (10)	1373 (3)	545 (26)
	178–197	429 (1)	683 (2)	1756 (4)	1625 (5)	1130 (12)
	198 & over	— (0)	15129 (1)	— (0)	2139 (3)	1703 (4)
	Total	279 (12)	566 (18)	1205 (19)	1542 (11)	614 (60)
40–49	157 & less	310 (10)	426 (17)	120 (1)	— (0)	344 (28)
	158–177	242 (12)	386 (21)	420 (15)	244 (3)	336 (51)
	178–197	326 (3)	260 (5)	754 (14)	1050 (10)	566 (32)
	198 & over	624 (1)	1298 (2)	691 (2)	1183 (10)	1035 (15)
	Total	280 (26)	391 (45)	489 (32)	741 (23)	414 (126)
50–59	157 & less	164 (6)	209 (8)	308 (2)	—* (–)	197 (16)
	158–177	156 (27)	296 (28)	442 (17)	314 (2)	237 (74)
	178–197	396 (19)	303 (14)	342 (15)	536 (5)	359 (53)
	198 & over	185 (1)	371 (4)	420 (6)	636 (12)	466 (23)
	Total	201 (53)	285 (54)	388 (40)	550 (19)	281 (166)
60 & over	157 & less	271 (3)	209 (1)	865 (1)	—* (–)	294 (5)
	158–177	179 (15)	348 (11)	268 (7)	409 (1)	236 (34)
	178–197	89 (3)	217 (6)	61 (1)	235 (1)	134 (11)
	198 & over	151 (1)	169 (2)	561 (3)	903 (6)	394 (12)
	Total	163 (22)	263 (20)	245 (12)	599 (8)	227 (62)
Total	157 & less	266 (26)	326 (32)	458 (9)	— (0)	309 (67)
	158–177	176 (58)	355 (69)	448 (49)	386 (9)	282 (185)
	178–197	279 (26)	281 (27)	420 (34)	802 (21)	364 (108)
	198 & over	218 (3)	372 (9)	473 (11)	876 (31)	559 (54)
	Total	212 (113)	332 (137)	441 (103)	708 (61)	327 (414)

* No entries.

TABLE 1(b)
AVERAGE OF CURRENT SYSTOLIC AND DIASTOLIC READINGS
Mortality Ratios (Actual Deaths in Parenthesis)
Women

Age	Systolic	Diastolic 5th Phase Reading: 97 & less	98–107	108–117	118 & over	Total
39 & less	157 & less	0% (0)	0% (0)	0% (0)	—% * (–)	0% (0)
	158–177	0 (0)	0 (0)	787 (1)	0 (0)	241 (1)
	178–197	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
	198 & over	— * (–)	— * (–)	— * (–)	0 (0)	0 (0)
	Total	0 (0)	0 (0)	514 (1)	0 (0)	136 (1)
40–49	157 & less	181 (2)	0 (0)	0 (0)	— * (–)	105 (2)
	158–177	0 (0)	168 (3)	113 (1)	0 (0)	73 (4)
	178–197	241 (1)	361 (3)	0 (0)	253 (1)	193 (5)
	198 & over	— * (–)	816 (1)	240 (1)	695 (4)	538 (6)
	Total	71 (3)	208 (7)	82 (2)	466 (5)	153 (17)
50–59	157 & less	250 (5)	173 (2)	252 (1)	— * (–)	225 (8)
	158–177	104 (8)	76 (3)	406 (4)	0 (0)	118 (15)
	178–197	172 (5)	120 (5)	0 (0)	464 (2)	131 (12)
	198 & over	0 (0)	0 (0)	0 (0)	250 (4)	87 (4)
	Total	133 (18)	98 (10)	120 (5)	282 (6)	130 (39)
60 & over	157 & less	165 (1)	0 (0)	— * (–)	— * (–)	123 (1)
	158–177	117 (5)	155 (2)	0 (0)	— * (–)	115 (7)
	178–197	184 (3)	203 (4)	168 (1)	0 (0)	184 (8)
	198 & over	133 (1)	479 (3)	204 (1)	136 (1)	230 (6)
	Total	138 (10)	220 (9)	123 (2)	112 (1)	159 (22)
Total	157 & less	215 (8)	94 (2)	164 (1)	— * (–)	171 (11)
	158–177	88 (13)	112 (8)	236 (6)	0 (0)	110 (27)
	178–197	180 (9)	172 (12)	31 (1)	275 (3)	154 (25)
	198 & over	59 (1)	240 (4)	96 (2)	308 (9)	191 (16)
	Total	123 (31)	145 (26)	119 (10)	288 (12)	142 (79)

* No entries.

TABLE 1(c)

AVERAGE OF CURRENT SYSTOLIC AND DIASTOLIC READINGS

Mortality Ratios (Actual Deaths in Parenthesis)

Men and Women

Age	Systolic	Diastolic 5th Phase Reading: 97 & less	98–107	108–117	118 & over	Total
39 & less	157 & less	394% (7)	364% (6)	1273% (5)	0% (0)	466% (18)
	158–177	164 (4)	610 (9)	1061 (11)	1298 (3)	520 (27)
	178–197	375 (1)	652 (2)	1476 (4)	1410 (5)	1001 (12)
	198 & over	0 (0)	15129 (1)	0 (0)	1937 (3)	1603 (4)
	Total	266 (12)	524 (18)	1129 (20)	1397 (11)	581 (61)
40–49	157 & less	277 (12)	369 (17)	98 (1)	0 (0)	299 (30)
	158–177	156 (12)	332 (24)	359 (16)	225 (3)	266 (55)
	178–197	300 (4)	291 (8)	500 (14)	816 (11)	449 (37)
	198 & over	624 (1)	1085 (3)	424 (3)	985 (14)	819 (21)
	Total	215 (29)	350 (52)	378 (34)	670 (28)	344 (143)
50–59	157 & less	195 (11)	201 (10)	287 (3)	— * (–)	205 (24)
	158–177	140 (35)	231 (31)	435 (21)	296 (2)	203 (89)
	178–197	311 (24)	217 (19)	251 (15)	490 (7)	272 (65)
	198 & over	68 (1)	200 (4)	231 (6)	459 (16)	282 (27)
	Total	178 (71)	220 (64)	311 (45)	447 (25)	230 (205)
60 & over	157 & less	233 (4)	145 (1)	865 (1)	— * (–)	238 (6)
	158–177	158 (20)	292 (13)	221 (7)	409 (1)	200 (41)
	178–197	120 (6)	211 (10)	90 (2)	172 (1)	152 (19)
	198 & over	141 (2)	276 (5)	391 (4)	500 (7)	319 (18)
	Total	154 (32)	248 (29)	214 (14)	404 (9)	204 (84)
Total	157 & less	252 (34)	285 (34)	388 (10)	0 (0)	278 (78)
	158–177	149 (71)	290 (77)	408 (55)	363 (9)	235 (212)
	178–197	245 (35)	235 (39)	310 (35)	647 (24)	290 (133)
	198 & over	130 (4)	318 (13)	295 (13)	619 (40)	388 (70)
	Total	183 (144)	276 (163)	356 (113)	571 (73)	270 (493)

* No entries.

TABLE 2(a)
HIGHEST SYSTOLIC OR DIASTOLIC ON EXAMINATION
Mortality Ratios (Actual Deaths in Parenthesis)
Men

Age	Systolic	Diastolic 5th Phase Reading: 97 & less	98–107	108–117	118 & over	Total
39 & less	157 & less	—%*(–)	235%(2)	866%(8)	0%(0)	557%(10)
	158–177	134 (3)	442 (8)	1119 (17)	761 (3)	520 (31)
	178–197	169 (1)	459 (2)	1582 (5)	1488 (5)	774 (13)
	198 & over	2589 (1)	8554 (1)	859 (1)	1870 (3)	1834 (6)
	Total	174 (5)	418 (13)	1078 (31)	1204 (11)	614 (60)
40–49	157 & less	0 (0)	338 (7)	230 (4)	0 (0)	296 (11)
	158–177	212 (9)	468 (30)	344 (18)	388 (6)	361 (63)
	178–197	396 (4)	263 (7)	578 (16)	649 (8)	456 (35)
	198 & over	624 (1)	1048 (2)	691 (2)	1234 (12)	1054 (17)
	Total	254 (14)	416 (46)	399 (40)	679 (26)	414 (126)
50–59	157 & less	— * (–)	219 (4)	147 (2)	— * (–)	188 (6)
	158–177	181 (26)	236 (29)	382 (19)	115 (1)	231 (75)
	178–197	374 (20)	244 (13)	379 (21)	581 (7)	350 (61)
	198 & over	133 (1)	346 (5)	323 (6)	636 (12)	404 (24)
	Total	230 (47)	244 (51)	349 (48)	504 (20)	281 (166)
60 & over	157 & less	865 (1)	0 (0)	704 (2)	— * (–)	541 (3)
	158–177	191 (15)	256 (10)	237 (5)	409 (1)	220 (31)
	178–197	85 (3)	241 (7)	153 (4)	352 (2)	166 (16)
	198 & over	151 (1)	97 (1)	586 (4)	903 (6)	394 (12)
	Total	165 (20)	225 (18)	263 (15)	609 (9)	227 (62)
Total	157 & less	476 (1)	280 (13)	371 (16)	0 (0)	324 (30)
	158–177	185 (53)	315 (77)	426 (59)	360 (11)	286 (200)
	178–197	267 (28)	256 (29)	409 (46)	659 (22)	343 (125)
	198 & over	248 (4)	335 (9)	441 (13)	896 (33)	540 (59)
	Total	210 (86)	297 (128)	414 (134)	648 (66)	327 (414)

* No entries.

TABLE 2(b)
HIGHEST SYSTOLIC OR DIASTOLIC ON EXAMINATION
Mortality Ratios (Actual Deaths in Parenthesis)
Women
Diastolic 5th Phase Reading

Age	Systolic	97 & less	98–107	108–117	118 & over	Total
39 & less	157 & less	—%*(–)	0%(0)	0%(0)	—%*(–)	0%(0)
	158–177	0 (0)	0 (0)	505 (1)	0 (0)	230 (1)
	178–197	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
	198 & over	— * (–)	— * (–)	— * (–)	0 (0)	0 (0)
	Total	0 (0)	0 (0)	346 (1)	0 (0)	136 (1)
40–49	157 & less	— * (–)	0 (0)	0 (0)	— * (–)	0 (0)
	158–177	37 (1)	125 (3)	123 (1)	0 (0)	82 (5)
	178–197	144 (1)	322 (3)	87 (1)	211 (1)	185 (6)
	198 & over	— * (–)	816 (1)	188 (1)	695 (4)	488 (6)
	Total	59 (2)	191 (7)	106 (3)	416 (5)	153 (17)
50–59	157 & less	— * (–)	0 (0)	202 (1)	— * (–)	84 (1)
	158–177	107 (7)	117 (6)	424 (6)	0 (0)	144 (19)
	178–197	211 (6)	102 (5)	0 (0)	404 (2)	126 (13)
	198 & over	90 (1)	0 (0)	0 (0)	305 (5)	114 (6)
	Total	133 (14)	92 (11)	133 (7)	311 (7)	130 (39)
60 & over	157 & less	— * (–)	0 (0)	— * (–)	— * (–)	0 (0)
	158–177	142 (6)	151 (2)	0 (0)	— * (–)	131 (8)
	178–197	183 (3)	173 (4)	168 (1)	0 (0)	170 (8)
	198 & over	100 (1)	573 (3)	204 (1)	119 (1)	210 (6)
	Total	146 (10)	206 (9)	123 (2)	101 (1)	159 (22)
Total	157 & less	— * (–)	0 (0)	114 (1)	— * (–)	48 (1)
	158–177	102 (14)	123 (11)	269 (8)	0 (0)	128 (33)
	178–197	192 (10)	147 (12)	52 (2)	256 (3)	147 (27)
	198 & over	95 (2)	208 (4)	88 (2)	326 (10)	192 (18)
	Total	124 (26)	134 (27)	130 (13)	288 (13)	142 (79)

* No entries.

TABLE 2(c)
HIGHEST SYSTOLIC OR DIASTOLIC ON EXAMINATION
Mortality Ratios (Actual Deaths in Parenthesis)
Men and Women

Age	Systolic	Diastolic 5th Phase Reading: 97 & less		98–107		108–117		118 & over		Total	
39 & less	157 & less	—%*	(–)	213%	(2)	824%	(8)	0%	(0)	518%	(10)
	158–177	124	(3)	431	(8)	1049	(18)	738	(3)	500	(32)
	178–197	160	(1)	431	(2)	1392	(5)	1306	(5)	710	(13)
	198 & over	2589	(1)	8554	(1)	859	(1)	1714	(3)	1755	(6)
	Total	162	(5)	398	(13)	1011	(32)	1114	(11)	581	(61)
40–49	157 & less	0	(0)	348	(7)	193	(4)	0	(0)	259	(11)
	158–177	144	(10)	374	(33)	314	(19)	353	(6)	289	(68)
	178–197	293	(5)	279	(10)	435	(17)	528	(9)	376	(41)
	198 & over	624	(1)	958	(3)	365	(3)	1034	(16)	809	(23)
	Total	180	(16)	360	(53)	335	(43)	616	(31)	344	(143)
50–59	157 & less	— *	(–)	158	(4)	162	(3)	— *	(–)	160	(7)
	158–177	158	(33)	201	(35)	391	(25)	102	(1)	206	(94)
	178–197	317	(26)	176	(18)	275	(21)	529	(9)	267	(74)
	198 & over	108	(2)	184	(5)	193	(6)	482	(17)	267	(30)
	Total	197	(61)	188	(62)	290	(55)	435	(27)	230	(205)
60 & over	157 & less	865	(1)	0	(0)	704	(2)	— *	(–)	393	(3)
	158–177	174	(21)	230	(12)	188	(5)	409	(1)	193	(39)
	178–197	116	(6)	211	(11)	156	(5)	276	(2)	167	(24)
	198 & over	120	(2)	257	(4)	427	(5)	466	(7)	305	(18)
	Total	158	(30)	218	(27)	232	(17)	405	(10)	204	(84)
Total	157 & less	476	(1)	223	(13)	328	(17)	0	(0)	273	(31)
	158–177	158	(67)	264	(88)	399	(67)	330	(11)	243	(233)
	178–197	242	(38)	210	(41)	318	(48)	554	(25)	277	(152)
	198 & over	161	(6)	282	(13)	287	(15)	637	(43)	379	(77)
	Total	181	(112)	245	(155)	347	(147)	537	(79)	270	(493)

* No entries.

TABLE 3(a)

AVERAGE OF TWO HIGHEST BLOOD PRESSURE READINGS WITHIN 5 YEARS

Mortality Ratios (Actual Deaths in Parenthesis)

Men

Age	Systolic	Diastolic 5th Phase Reading: 97 & less	98–107	108–117	118 & over	Total
39 & less	157 & less	336% (5)	428% (7)	917% (4)	—% (0)	446% (16)
	158–177	166 (4)	730 (11)	1062 (10)	1426 (3)	553 (28)
	178–197	406 (1)	1129 (3)	2822 (5)	1555 (4)	1374 (13)
	198 & over	—* (–)	—* (–)	— (0)	2457 (3)	1733 (3)
	Total	242 (10)	616 (21)	1183 (19)	1631 (10)	614 (60)
40–49	157 & less	443 (11)	386 (17)	121 (1)	— (0)	374 (29)
	158–177	246 (13)	415 (26)	465 (17)	107 (1)	353 (57)
	178–197	233 (2)	212 (4)	759 (15)	948 (7)	513 (28)
	198 & over	—* (–)	811 (2)	410 (1)	1499 (9)	1100 (12)
	Total	301 (26)	383 (49)	657 (44)	734 (17)	414 (126)
50–59	157 & less	62 (2)	339 (12)	227 (2)	—* (–)	210 (16)
	158–177	180 (32)	252 (26)	319 (12)	348 (2)	222 (72)
	178–197	411 (20)	313 (15)	383 (17)	725 (6)	389 (58)
	198 & over	—* (–)	438 (5)	366 (5)	609 (10)	482 (20)
	Total	209 (54)	293 (58)	345 (36)	591 (18)	281 (166)
60 & over	157 & less	308 (4)	209 (1)	865 (1)	—* (–)	317 (6)
	158–177	179 (15)	258 (11)	353 (7)	409 (1)	228 (34)
	178–197	105 (3)	172 (5)	69 (1)	235 (1)	131 (10)
	198 & over	221 (1)	144 (2)	791 (3)	903 (6)	416 (12)
	Total	177 (23)	210 (19)	306 (12)	599 (8)	227 (62)
Total	157 & less	259 (22)	368 (37)	354 (8)	— (0)	322 (67)
	158–177	189 (64)	331 (74)	445 (46)	356 (7)	279 (191)
	178–197	294 (26)	274 (27)	472 (38)	800 (18)	376 (109)
	198 & over	221 (1)	324 (9)	441 (9)	924 (28)	566 (47)
	Total	219 (113)	327 (147)	445 (101)	725 (53)	327 (414)

*No entries.

TABLE 3(b)

AVERAGE OF TWO HIGHEST BLOOD PRESSURE READINGS WITHIN 5 YEARS

Mortality Ratios (Actual Deaths in Parenthesis)

Women

Age	Systolic	Diastolic 5th Phase Reading: 97 & less	98–107	108–117	118 & over	Total
39 & less	157 & less	—% (0)	—% (0)	—% (0)	—%* (–)	—% (0)
	158–177	— (0)	— (0)	787 (1)	— (0)	228 (1)
	178–197	— (0)	— (0)	— (0)	— (0)	— (0)
	198 & over	—* (–)	—* (–)	—* (–)	— (0)	— (0)
	Total	— (0)	— (0)	542 (1)	— (0)	136 (1)
40–49	157 & less	205 (2)	— (0)	— (0)	—* (–)	135 (2)
	158–177	— (0)	151 (3)	100 (1)	— (0)	67 (4)
	178–197	241 (1)	354 (3)	— (0)	253 (1)	195 (5)
	198 & over	—* (–)	816 (1)	272 (1)	695 (4)	563 (6)
	Total	70 (3)	204 (7)	86 (2)	466 (5)	153 (17)
50–59	157 & less	130 (2)	152 (2)	252 (1)	—* (–)	154 (5)
	158–177	102 (8)	123 (5)	416 (4)	— (0)	132 (17)
	178–197	207 (6)	119 (5)	— (0)	404 (2)	140 (13)
	198 & over	—* (–)	— (0)	— (0)	250 (4)	88 (4)
	Total	130 (16)	105 (12)	121 (5)	282 (6)	130 (39)
60 & over	157 & less	165 (1)	— (0)	—* (–)	—* (–)	123 (1)
	158–177	117 (5)	136 (2)	— (0)	—* (–)	112 (7)
	178–197	184 (3)	203 (4)	242 (1)	— (0)	192 (8)
	198 & over	133 (1)	479 (3)	204 (1)	136 (1)	230 (6)
	Total	138 (10)	211 (9)	138 (2)	112 (1)	159 (22)
Total	157 & less	160 (5)	95 (2)	216 (1)	— (0)	140 (8)
	158–177	86 (13)	131 (10)	228 (6)	— (0)	113 (29)
	178–197	201 (10)	170 (12)	33 (1)	275 (3)	161 (26)
	198 & over	133 (1)	154 (4)	103 (2)	308 (9)	195 (16)
	Total	121 (29)	144 (28)	124 (10)	74 (12)	142 (79)

*No entries.

TABLE 3(c)

AVERAGE OF TWO HIGHEST BLOOD PRESSURE READINGS WITHIN 5 YEARS

Mortality Ratios (Actual Deaths in Parenthesis)

Men and Women

Age	Systolic	Diastolic 5th Phase Reading: 97 & less	98–107	108–117	118 & over	Total
39 & less	157 & less	334% (5)	399% (7)	868% (4)	—% (0)	428% (16)
	158–177	156 (4)	671 (11)	1029 (11)	1345 (3)	527 (29)
	178–197	357 (1)	1073 (3)	2376 (5)	1315 (4)	1210 (13)
	198 & over	—* (–)	—* (–)	— (0)	2194 (3)	1598 (3)
	Total	230 (10)	572 (21)	1117 (20)	1456 (10)	581 (61)
40–49	157 & less	376 (13)	349 (17)	115 (1)	—* (–)	336 (31)
	158–177	159 (13)	351 (29)	387 (18)	96 (1)	276 (61)
	178–197	236 (3)	256 (7)	520 (15)	706 (8)	411 (33)
	198 & over	—* (–)	813 (3)	327 (2)	1106 (13)	834 (18)
	Total	225 (29)	345 (56)	399 (36)	649 (22)	344 (143)
50–59	157 & less	84 (4)	289 (14)	234 (3)	—* (–)	193 (21)
	158–177	156 (40)	216 (31)	339 (16)	326 (2)	197 (89)
	178–197	335 (26)	222 (20)	278 (17)	604 (8)	293 (71)
	198 & over	—* (–)	167 (5)	204 (5)	432 (14)	276 (24)
	Total	184 (70)	272 (70)	281 (41)	464 (24)	230 (205)
60 & over	157 & less	262 (5)	145 (1)	865 (1)	—* (–)	258 (7)
	158–177	158 (20)	227 (13)	277 (7)	409 (1)	194 (41)
	178–197	134 (6)	184 (9)	107 (2)	172 (1)	152 (18)
	198 & over	166 (2)	248 (5)	460 (4)	500 (7)	328 (18)
	Total	163 (33)	210 (28)	260 (14)	404 (9)	204 (84)
Total	157 & less	232 (27)	321 (39)	330 (9)	— (0)	282 (75)
	158–177	157 (77)	280 (84)	401 (52)	330 (7)	234 (220)
	178–197	261 (36)	231 (39)	352 (39)	628 (21)	299 (135)
	198 & over	166 (2)	520 (13)	276 (11)	621 (37)	472 (63)
	Total	188 (142)	295 (175)	361 (111)	566 (65)	270 (493)

*No entries.

TABLE 4(a)

AVERAGE OF ALL SYSTOLIC AND DIASTOLIC READINGS WITHIN 5 YEARS

Mortality Ratios (Actual Deaths in Parenthesis)

Men

Age	Systolic	Diastolic 5th Phase Reading: 97 & less	98–107	108–117	118 & over	Total
39 & less	157 & less	337% (8)	494% (8)	866% (3)	—% (0)	435% (19)
	158–177	191 (4)	808 (10)	104 (9)	2380 (4)	619 (27)
	178–197	417 (1)	834 (2)	3256 (5)	1293 (3)	1271 (11)
	198 & over	—* (–)	—* (–)	— (0)	2457 (3)	1733 (3)
	Total	276 (13)	646 (20)	1200 (17)	1833 (10)	614 (60)
40–49	157 & less	317 (18)	435 (17)	132 (1)	— (0)	346 (36)
	158–177	268 (12)	419 (23)	447 (14)	116 (1)	358 (50)
	178–197	242 (2)	275 (5)	807 (14)	1094 (7)	558 (28)
	198 & over	624 (1)	1159 (1)	410 (1)	1609 (9)	1143 (12)
	Total	296 (33)	407 (46)	511 (30)	808 (17)	414 (126)
50–59	157 & less	129 (8)	303 (12)	419 (2)	—* (–)	207 (22)
	158–177	178 (31)	283 (25)	362 (12)	348 (2)	232 (70)
	178–197	381 (19)	318 (13)	361 (16)	725 (6)	377 (54)
	198 & over	214 (1)	665 (4)	366 (5)	642 (10)	501 (20)
	Total	203 (59)	309 (54)	365 (35)	608 (18)	281 (166)
60 & over	157 & less	258 (5)	— (0)	865 (1)	—* (–)	254 (6)
	158–177	170 (15)	298 (11)	353 (7)	409 (1)	231 (34)
	178–197	105 (3)	193 (5)	69 (1)	235 (1)	136 (10)
	198 & over	221 (1)	144 (2)	791 (3)	903 (6)	416 (12)
	Total	171 (24)	225 (18)	306 (12)	599 (8)	227 (62)
Total	157 & less	241 (39)	378 (37)	412 (7)	— (0)	299 (83)
	158–177	189 (62)	358 (69)	452 (42)	432 (8)	286 (181)
	178–197	280 (25)	286 (25)	464 (36)	800 (17)	374 (103)
	198 & over	278 (3)	337 (7)	441 (9)	965 (28)	580 (47)
	Total	219 (129)	346 (138)	452 (94)	763 (53)	327 (414)

* No entries.

TABLE 4(b)

AVERAGE OF ALL SYSTOLIC AND DIASTOLIC READINGS WITHIN 5 YEARS

Mortality Ratios (Actual Deaths in Parenthesis)

Women

Age	Systolic	Diastolic 5th Phase Reading: 97 & less	98–107	108–117	118 & over	Total
39 & less	157 & less	—%(0)	—%(0)	—%(0)	—%*(–)	—%(0)
	158–177	— (0)	— (0)	787 (1)	— (0)	254 (1)
	178–197	— (0)	— (0)	— (0)	— (0)	— (0)
	198 & over	—* (–)	—* (–)	—* (–)	— (0)	— (0)
	Total	— (0)	— (0)	542 (1)	— (0)	136 (1)
40–49	157 & less	160 (2)	— (0)	— (0)	—* (–)	106 (2)
	158–177	— (0)	220 (4)	— (0)	— (0)	72 (4)
	178–197	241 (1)	354 (3)	— (0)	253 (1)	195 (5)
	198 & over	—* (–)	816 (1)	272 (1)	695 (4)	563 (6)
	Total	68 (3)	240 (8)	44 (1)	466 (5)	153 (17)
50–59	157 & less	198 (4)	173 (2)	252 (1)	—* (–)	196 (7)
	158–177	101 (8)	96 (4)	416 (4)	— (0)	122 (16)
	178–197	168 (5)	126 (5)	— (0)	404 (2)	132 (12)
	198 & over	— (0)	— (0)	— (0)	250 (4)	93 (4)
	Total	122 (17)	110 (11)	128 (5)	282 (6)	130 (39)
60 & over	157 & less	136 (1)	— (0)	—* (–)	—* (–)	106 (1)
	158–177	121 (5)	136 (2)	— (0)	—* (–)	114 (7)
	178–197	184 (3)	203 (4)	242 (1)	— (0)	192 (8)
	198 & over	133 (1)	479 (3)	204 (1)	136 (1)	230 (6)
	Total	138 (10)	211 (9)	138 (2)	112 (1)	159 (22)
Total	157 & less	172 (7)	99 (2)	195 (1)	—* (–)	151 (10)
	158–177	87 (13)	132 (10)	198 (5)	— (0)	111 (28)
	178–197	178 (9)	176 (12)	34 (1)	275 (3)	157 (25)
	198 & over	57 (1)	273 (4)	110 (2)	308 (9)	201 (16)
	Total	116 (30)	157 (28)	115 (9)	288 (12)	142 (79)

*No entries.

TABLE 4(c)

AVERAGE OF ALL SYSTOLIC AND DIASTOLIC READINGS WITHIN 5 YEARS

Mortality Ratios (Actual Deaths in Parenthesis)

Men and Women

Age	Systolic	Diastolic 5th Phase Reading: 97 & less	98–107	108–117	118 & over	Total
39 & less	157 & less	329% (8)	461% (8)	809% (3)	—% (0)	416% (19)
	158–177	179 (4)	738 (10)	1008 (10)	2214 (4)	589 (28)
	178–197	366 (1)	788 (2)	2677 (5)	1076 (3)	1108 (11)
	198 & over	—* (–)	—* (–)	— (0)	2194 (3)	1598 (3)
	Total	263 (13)	598 (20)	1124 (18)	1614 (10)	581 (61)
40–49	157 & less	289 (20)	381 (17)	118 (1)	— (0)	309 (38)
	158–177	166 (12)	369 (27)	348 (14)	104 (1)	276 (54)
	178–197	242 (3)	300 (8)	530 (14)	773 (8)	435 (33)
	198 & over	624 (1)	958 (2)	327 (2)	1146 (13)	851 (18)
	Total	231 (36)	369 (54)	381 (31)	693 (22)	344 (143)
50–59	157 & less	146 (12)	274 (14)	343 (3)	—* (–)	204 (29)
	158–177	154 (39)	223 (29)	374 (16)	326 (2)	199 (86)
	178–197	301 (24)	223 (18)	266 (16)	604 (8)	282 (66)
	198 & over	68 (1)	303 (4)	215 (5)	444 (14)	290 (24)
	Total	177 (76)	236 (65)	296 (40)	471 (34)	230 (205)
60 & over	157 & less	224 (6)	— (0)	865 (1)	—* (–)	212 (7)
	158–177	154 (20)	252 (13)	277 (7)	409 (1)	196 (41)
	178–197	134 (6)	197 (9)	107 (2)	172 (1)	156 (18)
	198 & over	166 (2)	248 (5)	460 (4)	500 (7)	328 (18)
	Total	159 (34)	220 (27)	260 (14)	404 (9)	204 (84)
Total	157 & less	227 (46)	320 (39)	362 (8)	— (0)	271 (93)
	158–177	157 (75)	295 (79)	397 (47)	399 (8)	236 (209)
	178–197	243 (34)	238 (37)	345 (37)	621 (20)	294 (128)
	198 & over	141 (4)	310 (11)	285 (11)	635 (37)	392 (63)
	Total	187 (159)	287 (166)	360 (103)	585 (65)	270 (493)

* No entries.

CHART 1(a)

COMPARISON OF DIFFERENT METHODS OF UTILIZING BLOOD PRESSURES
(White Men, All Ages)

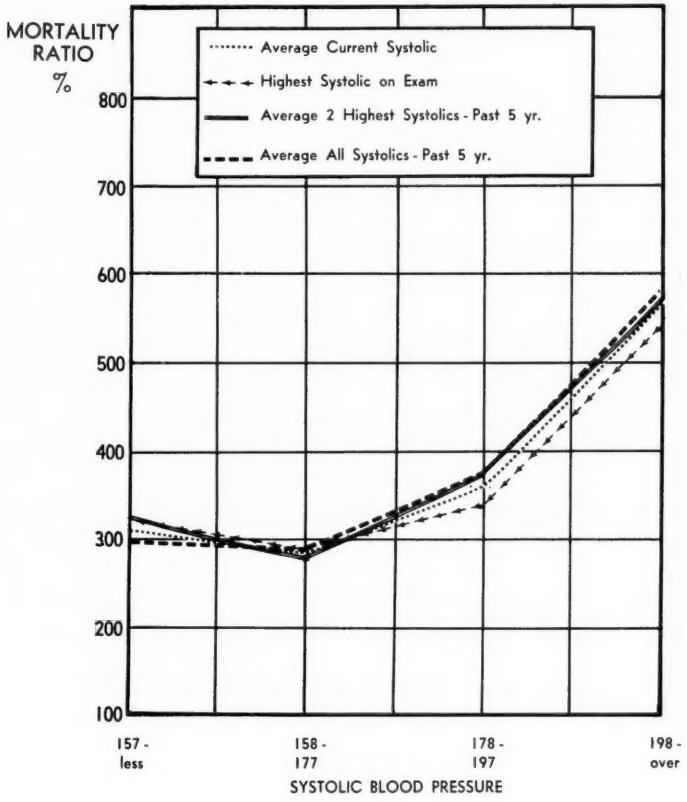
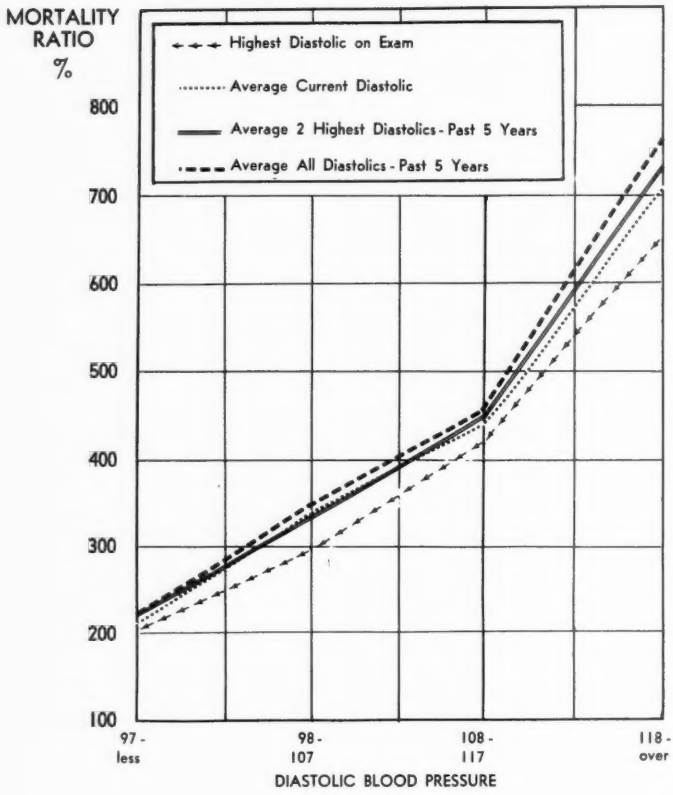


CHART 1(b)

COMPARISON OF DIFFERENT METHODS OF UTILIZING BLOOD PRESSURE

(White Men, All Ages)



tion, produce essentially the same trend and level of mortality ratios.

However, it should be pointed out that for a considerable proportion of cases only current blood pressure readings were available. Since these cases affect the results for all methods, it tends to mask somewhat the effect on mortality of using the highest reading or previous blood pressure readings derived from histories.

Chart 2(a) shows mortality ratios for groups of white men having about the same diastolic pressures but varying systolic. The results indicate that for men with similar diastolic pressures, variations in the systolic phase have limited effect on mortality. The one exception, namely, those with diastolic pressures of 118 mm. Hg and over, reflects the wide range of diastolics covered rather than any marked effect of the increase in systolic pressure.

On the other hand, an increase in diastolic pressure in men while holding the systolic pressure constant (chart 2(b)) shows a consistent increase in mortality.

No such increase in mortality with an increase in diastolic pressure is demonstrated in the limited data available on women.

Our data confirm the generally held clinical opinion that diastolic elevations are of greater importance than systolic in the prognosis of hypertensive disease.

Mean Blood Pressure

The statistical data were further analyzed using mean blood pressure as a basis for the mortality comparisons. The formula employed to obtain the mean pressure is diastolic reading plus one third of pulse pressure which is generally accepted as being satisfactory for clinical purposes.

In line with previously indicated findings, this formula gives greater weight to the diastolic pressure and is convenient since it permits the use of a single integer to reflect variations in the systolic and diastolic pressures.

To briefly indicate the ranges of blood pressures which the mean blood pressure figures represent, a few examples are shown in table 5.

CHART 2(a)

EFFECT OF INCREASE IN SYSTOLIC PRESSURE WITH CONSTANT DIASTOLIC PRESSURE

(White Men – All Ages)

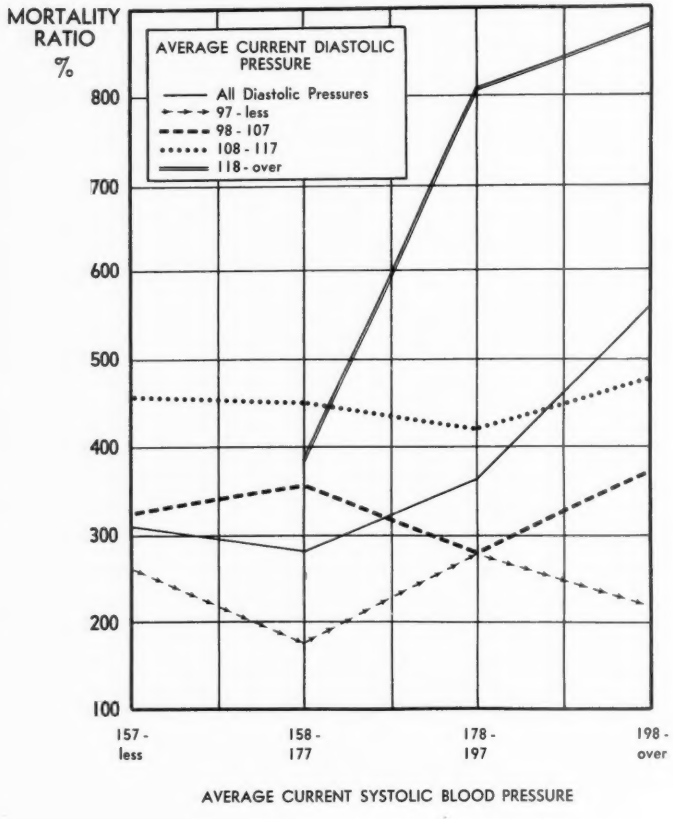
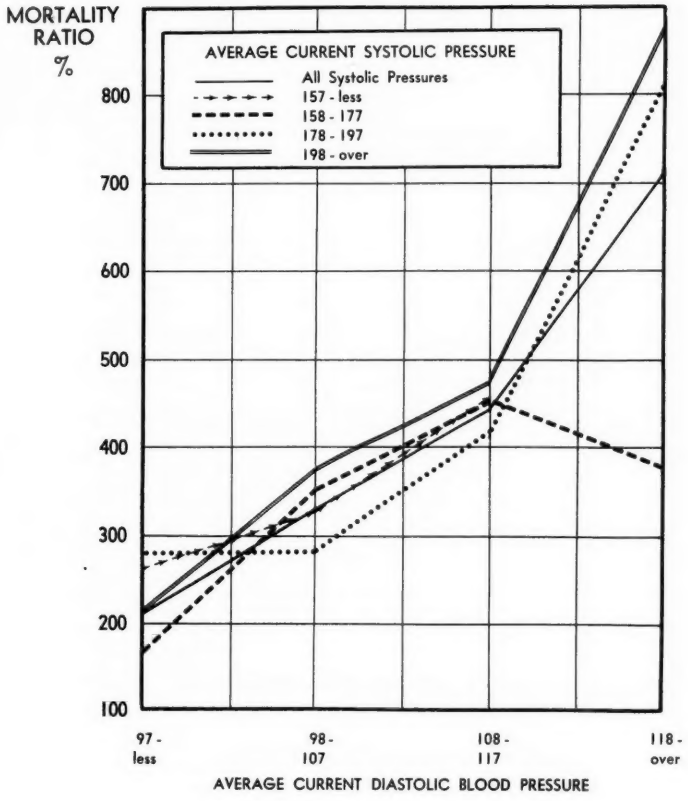


CHART 2(b)

EFFECT OF INCREASE IN DIASTOLIC PRESSURE WITH CONSTANT SYSTOLIC PRESSURE
(White Men – All Ages)



Subsequent aspects of this study combine mean blood pressures of 115 mm. Hg and less, e. g., below the range of 140/104, 164/92 and 190/79 mm. Hg. The remaining higher mean blood pressures

TABLE 5

Sample Blood Pressures (mm. Hg)				Mean Blood Pressure
140/89	160/79		=	106
140/104	164/92	190/79	=	116
160/109	190/94	210/84	=	126
170/119	200/104	212/98	=	136
190/124	200/119	212/114	=	146

represent individuals who are generally considered to have moderate to severe hypertension.

Table 6 and chart 3 show the mortality ratios obtained from the mean blood pressures based on the average of all current readings.

Sex

It is apparent from the previous tables and chart 4 that women with hypertension have a consistently lower mortality than do men of comparable age and blood pressure levels. The over-all mortality ratio of the men was 329 per cent, the women 142 per cent.

It should be pointed out that the basic table is based on a mixed insurance population of men and women. Considering men and women separately, when the basic table represents 100 per cent mortality, mortality for men would be roughly 105 per cent of this and that for women roughly 65 per cent. This would suggest that the over-all mortality ratio of 142 per cent for women in this study is actually 215 per cent as compared with standard mortality for this sex. Even this higher figure confirms the generally held opinion that women with hypertension have a better prognosis than similarly affected men.

Age

The previous tables and charts 4 and 5 show the variations in mortality by age at entry among hypertensives. The men consistently show a decrease in relative mortality as entry age increases. Women show a slight increase in relative mortality with an increase in age. Again we must point out that the data with respect to this sex are limited.

TABLE 6

MEAN BLOOD PRESSURE BASED ON AVERAGE OF CURRENT READINGS

Mortality Ratios (Actual Deaths in Parenthesis)

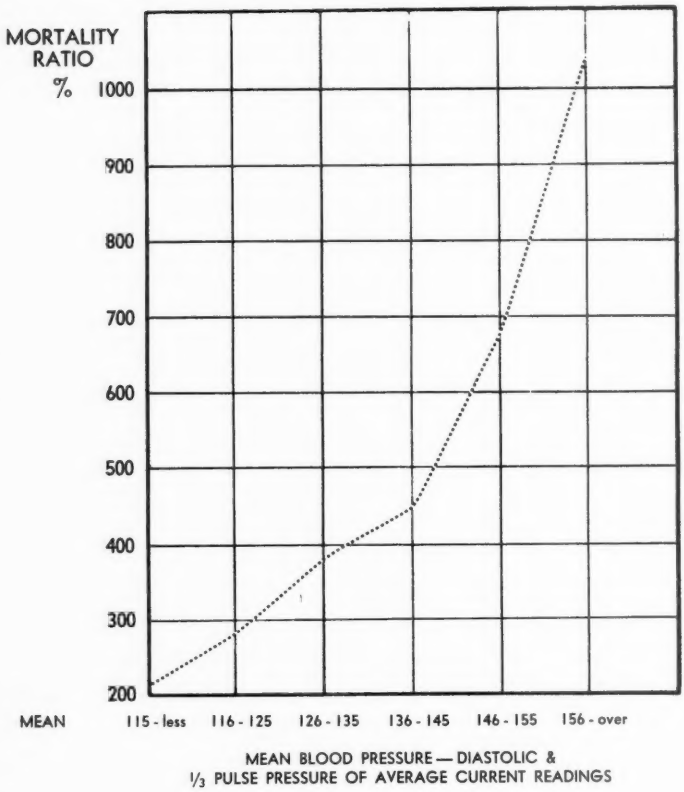
Age	Mean Blood Pressure	Men		Women		Men and Women	
39 & less	115 & less	254%	(9)	0%	(0)	248%	(9)
	116–125	573	(22)	0	(0)	521	(22)
	126–135	1030	(17)	666	(1)	999	(18)
	136–145	1530	(8)	0	(0)	1293	(8)
	146–155	1584	(2)	—*	(–)	1584	(2)
	156 & over	2286	(2)	0	(0)	1959	(2)
	Total	614	(60)	136	(1)	581	(61)
40–49	115 & less	239	(15)	76	(2)	191	(17)
	116–125	398	(48)	52	(2)	314	(50)
	126–135	396	(32)	257	(7)	361	(39)
	136–145	625	(18)	187	(2)	507	(20)
	146–155	1016	(8)	0	(0)	583	(8)
	156 & over	1440	(5)	1875	(4)	1606	(9)
	Total	414	(126)	153	(17)	344	(143)
50–59	115 & less	219	(29)	139	(8)	195	(37)
	116–125	222	(56)	121	(15)	189	(71)
	126–135	362	(47)	142	(10)	285	(57)
	136–145	375	(19)	39	(1)	262	(20)
	146–155	478	(8)	248	(3)	382	(11)
	156 & over	744	(7)	200	(2)	463	(9)
	Total	281	(166)	130	(39)	230	(205)
60 & over	115 & less	200	(13)	100	(4)	162	(17)
	116–125	178	(16)	146	(6)	168	(22)
	126–135	253	(20)	206	(7)	239	(27)
	136–145	202	(6)	231	(3)	211	(9)
	146–155	622	(5)	129	(1)	380	(6)
	156 & over	1315	(2)	336	(1)	668	(3)
	Total	227	(62)	159	(22)	204	(84)
All ages	115 & less	223	(66)	112	(14)	190	(80)
	116–125	284	(142)	111	(23)	233	(165)
	126–135	379	(116)	188	(25)	321	(141)
	136–145	446	(51)	119	(6)	346	(57)
	146–155	678	(23)	156	(4)	453	(27)
	156 & over	1047	(16)	459	(7)	753	(23)
	Total	327	(414)	142	(79)	270	(493)

*No entries.

CHART 3

EFFECT ON MORTALITY FROM INCREASING MEAN BLOOD PRESSURE

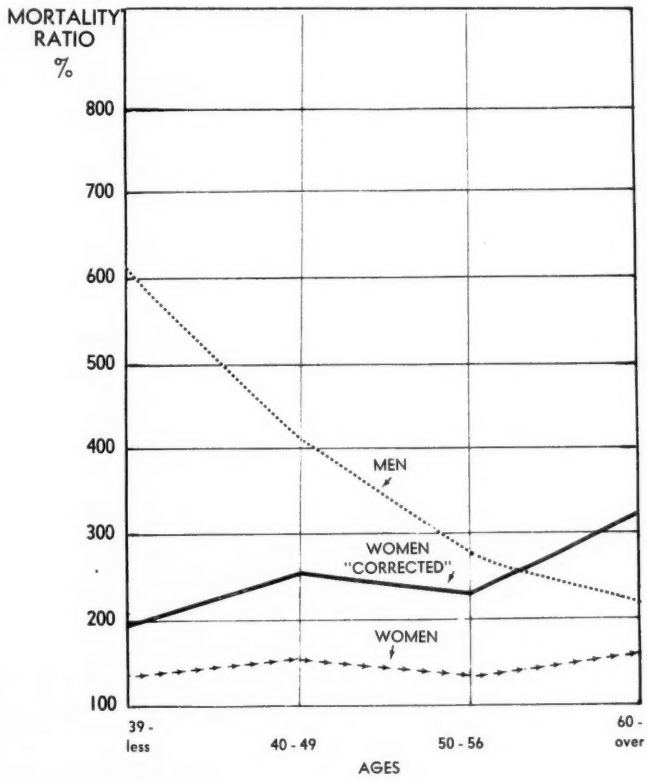
(White Men – All Ages)



The figures for men support the argument that higher pressures are more normal at the older ages. Therefore, more elevated levels than are currently acceptable might be within the range of insurability at the higher ages.

CHART 4

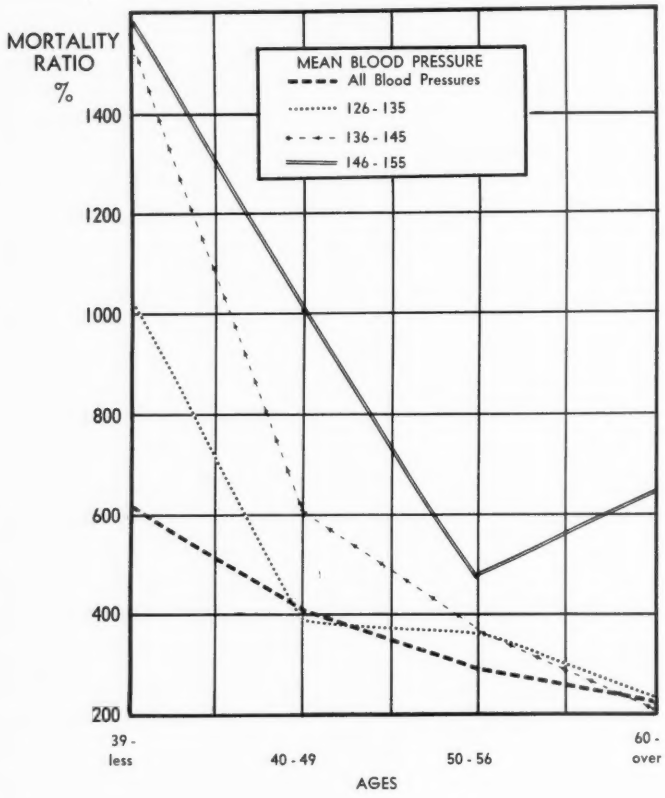
SEX DIFFERENCES IN MORTALITY BY AGE GROUPS IN WHITE HYPERTENSIVES



It is of interest that no reduction in the mortality with increasing age is apparent in women. When one adjusts the mortality for them as discussed previously (using appropriate corrections for each age group), it would appear that the mortality ratios of women and men above age 50 are about the same (chart 5).

CHART 5

EFFECT OF INCREASING AGE ON MORTALITY FROM HYPERTENSION
(White Men)



One may speculate from these curves that perhaps the popular idea that women are protected by some inherent factor against the effect of hypertension is not entirely valid. The decrease in mortality with increasing age in men might suggest some factor, per-

haps endocrine, rendering the younger ages of that sex more susceptible to the effects of hypertension. On the other hand, hypertension in these persons may be of an entirely different etiology, bringing about death at an earlier age.

Lability

Those men whose blood pressure fell below 140/90 mm. Hg on current examination, including rechecks, were found to have no excessive mortality (tables 7(a) and 7(b)).

TABLE 7(a)

EFFECT OF BLOOD PRESSURE LABILITY ON MORTALITY BASED ON CURRENT READINGS

Mortality Ratios (Actual Deaths in Parenthesis)

Men and Women

Age	Any Systolic Below 140 and any Diastolic Below 90 (mm. Hg)
39 & less	99%(2)
40–49	95 (5)
50–59	84 (6)
60–69	41 (1)
All ages	83 (14)

TABLE 7(b)

DISTRIBUTION OF ENTRANTS ACCORDING TO BLOOD PRESSURE LABILITY

Highest Current Blood Pressure Readings With at Least One Systolic Below 140 and One Diastolic Below 90 (mm. Hg)

Men and Women

Highest Systolic on Exam.	Highest Diastolic on Exam.				
	97 and less	98–107	108–117	118 & over	Total
157 & less	—*	37	27	—*	64
158–177	98	86	51	7	242
178–197	23	17	16	10	66
198 & over	1	2	5	1	9
Total	122	142	99	18	381

*No entries.

TABLE 8(a)

EFFECT OF BUILD ON MORTALITY
MORTALITY RATIOS (ACTUAL DEATHS IN PARENTHESIS)

Men

Age	Basic Rating	Mean Blood Pressure of Current Readings: 115 & Less	116–125	126–135	136–145	146–155	156 & Over	Total
39 & less	115 & less	281% (7)	680% (17)	1047% (10)	2008% (6)	4019% (2)	2392% (1)	679% (43)
	120–125	279 (1)	751 (2)	1341 (2)	4392 (1)	— (0)	—* (–)	731 (6)
	130	654 (1)	— (0)	1685 (1)	—* (–)	—* (–)	— (0)	603 (2)
	135	— (0)	551 (1)	2471 (2)	— (0)	— (0)	—* (–)	690 (3)
	140	— (0)	— (0)	— (0)	— (0)	— (0)	11038 (1)	378 (1)
	145	— (0)	— (0)	2895 (1)	—* (–)	—* (–)	—* (–)	470 (1)
	150	— (0)	— (0)	— (0)	— (0)	—* (–)	— (0)	— (0)
	155 & over	— (0)	360 (2)	385 (1)	1353 (1)	— (0)	—* (–)	337 (4)
	Total	254 (9)	573 (22)	1030 (17)	1530 (8)	1584 (2)	2286 (2)	614 (60)
40–49	115 & less	244 (12)	410 (35)	296 (18)	689 (13)	1178 (6)	1274 (3)	392 (87)
	120–125	184 (1)	297 (4)	823 (6)	350 (1)	—* (–)	2764 (1)	442 (13)
	130	1739 (2)	1021 (2)	1150 (2)	—* (–)	— (0)	— (0)	1085 (6)
	135	— (0)	369 (1)	936 (1)	718 (1)	— (0)	— (0)	399 (3)
	140	—* (–)	1094 (2)	— (0)	— (0)	—* (–)	—* (–)	581 (2)
	145	— (0)	363 (1)	737 (1)	— (0)	— (0)	7886 (1)	439 (3)
	150	— (0)	— (0)	717 (1)	—* (–)	— (0)	—* (–)	204 (1)
	155 & over	— (0)	297 (3)	503 (3)	704 (3)	1574 (2)	—* (–)	440 (11)
	Total	239 (15)	398 (48)	396 (12)	625 (18)	1016 (8)	1440 (5)	414 (126)

50–59	115 & less	258	(27)	229	(45)	397	(41)	473	(17)	484	(7)	741	(4)	307	(141)
	120–125	88	(1)	224	(6)	209	(3)	248	(1)	705	(1)	—	(0)	200	(12)
	130	—	(0)	298	(1)	—	(0)	—	(0)	—*	(–)	5426	(1)	223	(2)
	135	—	(0)	—	(0)	760	(1)	—	(0)	—	(0)	15848	(1)	183	(2)
	140	—	(0)	543	(2)	259	(1)	—	(0)	—*	(–)	3169	(1)	334	(4)
	145	—*	(–)	—	(0)	—	(0)	—	(0)	—*	(–)	—*	(–)	—	(0)
	150	—*	(–)	—	(0)	—*	(–)	—*	(–)	—*	(–)	—	(0)	—	(0)
	155 & over	118	(1)	132	(2)	233	(1)	524	(1)	—*	(–)	—	(0)	164	(5)
	Total	219	(29)	222	(56)	362	(47)	375	(19)	478	(8)	744	(7)	281	(166)
60 & over	115 & less	200	(12)	165	(11)	254	(17)	282	(6)	622	(5)	1121	(1)	232	(52)
	120–125	865	(1)	297	(4)	203	(4)	—	(0)	—*	(–)	1592	(1)	258	(8)
	130	—	(0)	—*	(–)	—	(0)	—*	(–)	—*	(–)	—*	(–)	—	(0)
	135	—*	(–)	—*	(–)	—*	(–)	—*	(–)	—*	(–)	—*	(–)	—*	(–)
	140	—*	(–)	—*	(–)	1121	(1)	—	(0)	—*	(–)	—*	(–)	288	(1)
	145	—*	(–)	—*	(–)	—*	(–)	—*	(–)	—*	(–)	—*	(–)	—*	(–)
	150	—	(0)	—	(0)	—*	(–)	—*	(–)	—*	(–)	—*	(–)	—	(0)
	155 & over	—*	(–)	135	(1)	—*	(–)	—*	(–)	—*	(–)	—*	(–)	135	(1)
	Total	200	(13)	178	(16)	253	(20)	202	(6)	622	(5)	1315	(2)	227	(62)
All ages	115 & less	243	(58)	289	(108)	357	(86)	531	(42)	712	(20)	993	(9)	333	(323)
	120–125	186	(4)	284	(16)	394	(13)	231	(3)	605	(1)	648	(2)	303	(39)
	130	510	(3)	471	(3)	588	(3)	—	(0)	—	(0)	1468	(1)	467	(10)
	135	—	(0)	265	(2)	1252	(4)	249	(1)	—	(0)	3091	(1)	351	(8)
	140	—	(0)	662	(4)	296	(2)	—	(0)	—*	(–)	4924	(2)	372	(8)
	145	—	(0)	220	(1)	681	(2)	—	(0)	—	(0)	7886	(1)	290	(4)
	150	—	(0)	—	(0)	586	(1)	—	(0)	—	(0)	—	(0)	70	(1)
	155 & over	69	(1)	210	(8)	389	(5)	724	(5)	1209	(2)	—	(0)	281	(21)
	Total	223	(66)	284	(142)	379	(116	446	(51)	678	(23)	1047	(16)	327	(414)

*No entries.

TABLE 8(b)

EFFECT OF BUILD ON MORTALITY
MORTALITY RATIOS (ACTUAL DEATHS IN PARENTHESIS)

Men and Women

Age	Basic Rating	Mean Blood Pressure of Current Readings: 115 & Less	116–125	126–135	136–145	146–155	156 & Over	Total
39 & less	115 & less	276% (7)	627% (17)	928% (10)	1673% (6)	4019% (2)	1772% (1)	634% (43)
	120–125	279 (1)	641 (2)	1341 (2)	2828 (1)	— (0)	—* (–)	683 (6)
	130	654 (1)	— (0)	1685 (1)	—* (–)	—* (–)	— (0)	603 (2)
	135	— (0)	521 (1)	2471 (2)	— (0)	— (0)	—* (–)	674 (3)
	140	— (0)	— (0)	— (0)	— (0)	—* (–)	11038 (1)	347 (1)
	145	— (0)	— (0)	2895 (1)	—* (–)	—* (–)	—* (–)	423 (1)
	150	— (0)	— (0)	2637 (1)	— (0)	—* (–)	— (0)	534 (1)
	155 & over	— (0)	310 (2)	356 (1)	1030 (1)	— (0)	—* (–)	292 (4)
	Total	248 (9)	521 (22)	999 (18)	1293 (8)	1584 (2)	1959 (2)	581 (61)
40–49	115 & less	200 (14)	328 (37)	279 (23)	541 (14)	689 (6)	1361 (6)	329 (100)
	120–125	114 (1)	231 (4)	696 (7)	549 (2)	— (0)	4511 (2)	393 (16)
	130	1739 (2)	539 (2)	864 (2)	—* (–)	— (0)	— (0)	666 (6)
	135	— (0)	369 (1)	936 (1)	718 (1)	— (0)	— (0)	399 (3)
	140	— (0)	1094 (2)	— (0)	— (0)	—* (–)	—* (–)	474 (2)
	145	— (0)	316 (1)	737 (1)	— (0)	— (0)	7886 (1)	358 (3)
	150	— (0)	— (0)	554 (1)	— (0)	— (0)	—* (–)	135 (1)
	155 & over	— (0)	208 (3)	526 (4)	510 (3)	1574 (2)	—* (–)	356 (12)
Total	191 (17)	314 (50)	361 (39)	507 (20)	583 (8)	1606 (9)	344 (143)	

50–59	115 & less	230	(33)	204	(58)	289	(46)	318	(18)	387	(10)	490	(5)	250	(170)
	120–125	94	(2)	162	(7)	185	(4)	205	(1)	705	(1)	—	(0)	155	(15)
	130	—	(0)	153	(1)	1042	(2)	—	(0)	—*	(–)	5426	(1)	269	(4)
	135	—	(0)	—	(0)	1256	(2)	—	(0)	—	(0)	15848	(1)	221	(3)
	140	—	(0)	344	(2)	184	(1)	—	(0)	—*	(–)	644	(1)	237	(4)
	145	—*	(–)	—	(0)	488	(1)	—	(0)	—*	(–)	—*	(–)	159	(1)
	150	—	(0)	—	(0)	—*	(–)	—*	(–)	—*	(–)	—	(0)	—	(0)
	155 & over	147	(2)	113	(3)	119	(1)	167	(1)	—	(0)	383	(1)	138	(8)
	Total	195	(37)	189	(71)	285	(57)	262	(20)	382	(11)	463	(9)	230	(205)
60 & over	115 & less	148	(14)	141	(14)	258	(24)	258	(8)	407	(6)	1121	(1)	201	(67)
	120–125	776	(2)	309	(5)	155	(2)	—	(0)	—*	(–)	865	(2)	276	(11)
	130	—	(0)	—	(0)	—	(0)	—*	(–)	—*	(–)	—*	(–)	—	(0)
	135	—	(0)	—*	(–)	—	(0)	600	(1)	—*	(–)	—*	(–)	189	(1)
	140	—*	(–)	—	(0)	459	(1)	—	(0)	—*	(–)	—*	(–)	173	(1)
	145	—*	(–)	933	(1)	—*	(–)	—	(0)	—*	(–)	—*	(–)	381	(1)
	150	518	(1)	—	(0)	—*	(–)	—*	(–)	—	(0)	—*	(–)	181	(1)
	155 & over	—	(0)	249	(2)	—	(0)	—*	(–)	—*	(–)	—	(0)	158	(2)
	Total	162	(17)	168	(22)	239	(27)	211	(9)	380	(6)	668	(3)	204	(84)
All ages	115 & less	204	(68)	241	(126)	298	(103)	393	(46)	482	(24)	809	(13)	274	(380)
	120–125	165	(6)	226	(18)	325	(15)	271	(4)	466	(1)	576	(4)	258	(48)
	130	368	(3)	212	(3)	817	(5)	—	(0)	—	(0)	1468	(1)	357	(12)
	135	—	(0)	219	(2)	946	(5)	352	(2)	—	(0)	3091	(1)	324	(10)
	140	—	(0)	424	(4)	200	(2)	—	(0)	—*	(–)	1218	(2)	269	(8)
	145	—	(0)	290	(2)	800	(3)	—	(0)	—	(0)	7886	(1)	305	(6)
	150	338	(1)	—	(0)	916	(2)	—	(0)	—	(0)	—	(0)	150	(3)
	155 & over	88	(2)	181	(10)	290	(6)	390	(5)	843	(2)	257	(1)	221	(26)
	Total	190	(80)	233	(165)	321	(141)	346	(57)	453	(27)	753	(23)	270	(493)

*No entries.

TABLE 9(a)
EFFECT OF EKG ON MORTALITY
MORTALITY RATIOS (ACTUAL DEATHS IN PARENTHESIS)
Men
Mean Blood Pressure of Average Current Readings

Age	EKG	115 & less	116–125	126–135	136–145	146–155	156 & over	Total
39 & less	No EKG	290% (9)	565% (20)	1062% (16)	1429% (7)	1949% (2)	2286% (2)	634% (56)
	Normal	— (0)	717 (2)	692 (1)	3013 (1)	— (0)	—* (–)	441 (4)
	Abnormal	— (0)	— (0)	—* (–)	—* (–)	—* (–)	—* (–)	— (0)
	Total	254 (9)	573 (22)	1030 (17)	1530 (8)	1584 (2)	2286 (2)	614 (60)
40–49	No EKG	238 (13)	432 (46)	411 (31)	647 (18)	1016 (8)	1440 (5)	439 (121)
	Normal	278 (2)	153 (2)	— (0)	— (0)	—* (–)	—* (–)	157 (4)
	Abnormal	— (0)	— (0)	950 (1)	— (0)	—* (–)	—* (–)	309 (1)
	Total	239 (15)	398 (48)	396 (32)	625 (118)	1016 (8)	1440 (5)	414 (126)
50–59	No EKG	224 (27)	241 (55)	375 (47)	412 (19)	439 (7)	744 (7)	297 (162)
	Normal	87 (1)	47 (1)	— (0)	— (0)	1242 (1)	—* (–)	75 (3)
	Abnormal	7924 (1)	— (0)	— (0)	— (0)	—* (–)	—* (–)	189 (1)
	Total	219 (29)	222 (56)	362 (47)	375 (19)	478 (8)	744 (7)	281 (166)
60 & over	No EKG	204 (11)	184 (16)	250 (19)	213 (6)	622 (5)	1315 (2)	232 (159)
	Normal	146 (1)	— (0)	— (0)	— (0)	—* (–)	—* (–)	78 (1)
	Abnormal	247 (1)	—* (–)	513 (1)	—* (–)	—* (–)	—* (–)	333 (2)
	Total	200 (13)	178 (16)	253 (20)	202 (6)	622 (5)	1315 (2)	227 (62)
All ages	No EKG	230 (60)	300 (137)	387 (113)	468 (50)	669 (22)	1047 (16)	342 (398)
	Normal	134 (4)	124 (5)	94 (1)	182 (1)	960 (1)	—* (–)	137 (12)
	Abnormal	381 (2)	— (0)	535 (2)	— (0)	— (0)	—* (–)	270 (4)
	Total	223 (66)	284 (142)	379 (116)	446 (51)	678 (23)	1047 (16)	327 (414)

*No entries.

TABLE 9(b)

EFFECT OF EKG ON MORTALITY
MORTALITY RATIOS (ACTUAL DEATHS IN PARENTHESIS)

Men and Women

(No Deaths Among Women in this Group)

Mean Blood Pressure Based on Average of Current Readings

Age	EKG	115 & less	116–125	126–135	136–145	146–155	156 & over	Total
39 & less	No EKG	281% (9)	512% (20)	1026% (17)	1196% (7)	1949% (2)	1959% (2)	597% (57)
	Normal	— (0)	682 (2)	692 (1)	3013 (1)	— (0)	—* (–)	434 (4)
	Abnormal	— (0)	— (0)	—* (–)	—* (–)	—* (–)	—* (–)	— (0)
	Total	248 (9)	521 (22)	999 (18)	1293 (8)	1584 (2)	1959 (2)	581 (61)
40–49	No EKG	189 (15)	333 (48)	372 (38)	527 (20)	583 (8)	1606 (9)	360 (138)
	Normal	233 (2)	141 (2)	— (0)	— (0)	—* (–)	—* (–)	141 (4)
	Abnormal	— (0)	— (0)	950 (1)	— (0)	—* (–)	—* (–)	263 (1)
	Total	191 (17)	314 (50)	361 (39)	507 (20)	583 (8)	1606 (9)	344 (143)
50–59	No EKG	200 (35)	200 (70)	292 (57)	279 (20)	357 (10)	463 (9)	239 (201)
	Normal	69 (1)	43 (1)	— (0)	— (0)	1242 (1)	—* (–)	67 (3)
	Abnormal	7924 (1)	— (0)	— (0)	— (0)	—* (–)	—* (–)	166 (1)
	Total	195 (37)	189 (71)	285 (57)	262 (20)	382 (11)	463 (9)	230 (205)
60 & over	No EKG	160 (15)	172 (22)	236 (26)	219 (9)	380 (6)	668 (3)	206 (81)
	Normal	146 (1)	— (0)	— (0)	— (0)	—* (–)	—* (–)	78 (1)
	Abnormal	247 (1)	—* (–)	513 (1)	—* (–)	—* (–)	—* (–)	333 (2)
	Total	162 (17)	168 (22)	239 (27)	211 (9)	380 (6)	668 (3)	204 (84)
All ages	No EKG	194 (74)	242 (160)	326 (138)	357 (56)	444 (26)	753 (23)	279 (477)
	Normal	117 (4)	115 (5)	91 (1)	182 (1)	960 (1)	—* (–)	126 (12)
	Abnormal	381 (2)	— (0)	447 (2)	— (0)	— (0)	—* (–)	248 (4)
	Total	190 (80)	233 (165)	321 (141)	346 (57)	453 (27)	753 (23)	270 (493)

TABLE 10(a)

EFFECT OF HEART SIZE BY CHEST X-RAY
MORTALITY RATIOS (ACTUAL DEATHS IN PARENTHESIS)

Men

Age	X-Ray	Mean Blood Pressure Based on Average of Current Readings: 115 & less	116–125	126–135	136–145	146–155	156 & over	Total
39 & less	No x-ray	282% (9)	551% (20)	1037% (16)	1602% (8)	2272% (2)	2286% (2)	630% (57)
	Normal	— (0)	1121 (2)	931 (1)	— (0)	— (0)	—* (–)	443 (3)
	Total	254 (9)	573 (22)	1030 (17)	1530 (8)	1584 (2)	2286 (2)	614 (60)
40–49	No x-ray	232 (13)	427 (46)	409 (31)	618 (17)	1016 (8)	1440 (5)	431 (120)
	Normal	345 (2)	169 (2)	214 (1)	784 (1)	—* (–)	—* (–)	255 (6)
	Total	239 (15)	398 (48)	396 (32)	625 (18)	1016 (8)	1440 (5)	414 (126)
50–59	No x-ray	223 (27)	250 (56)	371 (47)	400 (19)	478 (8)	744 (7)	300 (164)
	Normal	200 (2)	— (0)	— (0)	— (0)	—* (–)	—* (–)	50 (2)
	Total	219 (29)	222 (56)	362 (47)	375 (19)	478 (8)	744 (7)	281 (166)
60 & over	No x-ray	204 (11)	188 (16)	259 (20)	213 (6)	622 (5)	1315 (2)	236 (60)
	Normal	184 (2)	— (0)	— (0)	—* (–)	—* (–)	—* (–)	127 (2)
	Total	200 (13)	178 (16)	253 (20)	202 (6)	622 (5)	1315 (2)	227 (62)
All ages	No x-ray	228 (60)	304 (138)	386 (114)	463 (50)	686 (23)	1047 (16)	343 (401)
	Normal	200 (6)	96 (4)	210 (2)	220 (1)	— (0)	— (0)	151 (13)
	Total	223 (66)	284 (142)	379 (116)	446 (51)	678 (23)	1047 (16)	327 (414)

Note: In the entire series there were 20 cases with abnormal chest x-rays but no deaths occurred among these.
*No entries.

TABLE 10(b)

EFFECT OF HEART SIZE BY CHEST X-RAY
MORTALITY RATIOS (ACTUAL DEATHS IN PARENTHESIS)

Men and Women

		Mean Blood Pressure Based on Average of Current Readings											
Age	**X-Ray**	**115 & less**		**116–125**		**126–135**		**136–145**		**146–155**		**156 & over**		**Total**	
39 & less	No x-ray	274%	(9)	500%	(20)	1004%	(17)	1345%	(8)	2272%	(2)	1959%	(2)	594%	(58)
	Normal	—	(0)	1121	(2)	931	(1)	—	(0)	—	(0)	—*	(–)	443	(3)
	Total	248	(9)	521	(22)	999	(18)	1293	(8)	1584	(2)	1959	(2)	581	(61)
40–49	No x-ray	185	(15)	331	(48)	370	(38)	505	(19)	583	(8)	1606	(9)	355	(137)
	Normal	278	(2)	161	(2)	198	(1)	540	(1)	—*	(–)	—*	(–)	226	(6)
	Total	191	(17)	314	(50)	361	(39)	507	(20)	583	(8)	1606	(9)	344	(143)
50–59	No x-ray	199	(35)	205	(71)	290	(57)	274	(20)	382	(11)	463	(9)	242	(203)
	Normal	155	(2)	—	(0)	—	(0)	—	(0)	—*	(–)	—*	(–)	44	(2)
	Total	195	(37)	189	(71)	285	(57)	262	(20)	382	(11)	463	(9)	230	(205)
60 & over	No x-ray	160	(15)	174	(22)	247	(27)	219	(9)	380	(6)	668	(3)	210	(82)
	Normal	184	(2)	—	(0)	—	(0)	—	(0)	—*	(–)	—*	(–)	115	(2)
	Total	162	(17)	168	(22)	239	(27)	211	(9)	380	(6)	668	(3)	204	(84)
All ages	No x-ray	193	(74)	245	(161)	327	(139)	355	(56)	456	(27)	753	(23)	280	(480)
	Normal	175	(6)	91	(4)	163	(2)	195	(1)	—	(0)	—*	(–)	136	(13)
	Total	190	(80)	233	(165)	321	(141)	346	(57)	453	(27)	753	(23)	270	(493)

Note: In the entire series there were 20 cases with abnormal chest x-rays but no deaths occurred among these.

Other methods of studying lability resulted in contradictory results and it is felt that further study is indicated before any definitive conclusion can be drawn.

Build

No consistent effect of build on the mortality of hypertensive individuals in any age group was noted (tables 8(a) and 8(b)).

Electrocardiograms

There were electrocardiograms on 231 individuals of which 23 were abnormal. Tables 9(a) and 9(b) show the effect of normal electrocardiograms as compared to abnormal or no electrocardiogram. The mortality ratio for men with no electrocardiograms was 342 per cent, with normal electrocardiograms 137 per cent, and with abnormal electrocardiograms 270 per cent.

The tables show that in the group with lower mean pressures, those with abnormal electrocardiograms had a greater mortality than those with no electrocardiogram. Furthermore, the excessive mortality in those with no electrocardiogram is due to the results in the higher blood pressure ranges. Since electrocardiograms are not usually requested in cases where the high level of the blood pressure justified prompt rejection of the risk, it is not surprising that this group would show the greatest mortality.

Heart Size by X-ray

Two hundred and eight chest x-rays were studied. Only 20 x-rays with cardiomegaly were found and in this group no individuals died. The mortality ratio of individuals with normal heart size was 136 per cent against 280 per cent for those with no chest x-rays (tables 10(a) and 10(b)).

We feel that the satisfactory mortality found in individuals with a normal electrocardiogram and chest x-ray indicates the uncomplicated nature of their hypertension. Hence, we believe that these procedures are important in the precise evaluation of the risk in hypertension.

Conclusions

This study comprised 3,283 individuals with blood pressures exceeding 162 mm. Hg systolic and/or 102 mm. Hg fifth phase diastolic, who applied for life insurance between 1946 and 1951. The following conclusions were drawn:

1. In evaluating the prognosis of a hypertensive male individual, greater weight must be placed on the diastolic elevation than on systolic.

2. For the purpose of obtaining a single integer in the utilization of blood pressure readings, a mean blood pressure (diastolic plus one third of pulse pressure) appears to give consistent results.

3. Women with hypertension show only a slight increase in mortality with increasing blood pressure levels, and a considerably better mortality than do similarly affected men up to age 50.

4. The relative mortality from hypertension in men declines as the age of entry advances, approaching that of women at age 50 and above. The mortality of women increases slightly with age. This suggests the possibility of some additional factor being responsible for the higher mortality in men with hypertension under age 50.

5. Our investigation of the effect of blood pressure lability on mortality yields conflicting results and no definite conclusions can be drawn without further study. However, the very favorable mortality demonstrated in cases where the blood pressure dropped to entirely normal levels may be of significance.

6. Electrocardiograms and chest x-rays appear to provide effective screening for hypertensives and, when normal, a more favorable mortality may be expected.

7. Obesity does not appear to be a significant additive factor in the prognosis of hypertensives within the range studied.

PRESIDENT GETMAN — Thank you very much, Dr. Harnes. We are now ready for questions and will take any questions that you want to ask Dr. Pollack first.

DR. HARRY E. UNGERLEIDER — I do not have any questions, but I am impelled to make a comment here, because I think that it should be made.

These two papers, at least to my mind, should mark the death knell of what is known as "intercompany studies". In intercompany studies, we have a mass of heterogeneous blood pressure statistics that go through some sort of actuarial or mathematical manipulation resulting generally in the observations "with each quinquennial increase in the systolic pressure, there is an increase in mortality". These two papers do not confirm that.

We also find that when we look into the source of material from the various companies, the particular material furnished is anything but homogeneous. Here, we have two excellent papers with medical supervision, scientifically put together, and I would like to say at this particular moment that this ought to be encouraged. There should be more individual company studies, under the direction of doctors, set up as these papers have been.

I was particularly impressed and pleased to see certain confirmations of previous work reported in this organization, for example, the effects of the electrocardiogram in the assessment of blood pressure. Years ago, we made a similar study on groups of the same build, age, and all other characteristics, the only variable being the electrocardiogram and the blood pressure under observation being constant at 160/100 mm. Hg. We had relatively 300 cases with normal electrocardiograms, and the blood pressure was 160/100 mm. Hg. With normal electrocardiogram, our mortality was 184 per cent. With what we call a questionable electrocardiogram, not exactly normal, the mortality increased from 184 per cent to somewhere around 250 per cent. With a pattern of left ventricular hypertrophy, the mortality went up to about 375 per cent. And, of course, with bundle branch block and other deviations of the electrocardiogram, it was somewhere around 500 per cent. This was the experience of one company only.

I would certainly ask each and every one of you who can do so to make mortality studies of each and every impairment where the material is available.

These two papers deserve the commendation of our entire Association.

PRESIDENT GETMAN — Are there any other questions?

DR. PAUL H. LANGNER, JR. — Correct me if I am wrong, but I believe he said no difference was found between labile and nonlabile blood pressures in the mortality ratios. Then Dr. Harnes said that, in labile blood pressure, if it fell below 140/90 on a current examination, there was no increase in mortality.

I wonder if there are any conflicts between these two statements or perhaps Dr. Pollack found the same thing in labile pressures when they went really low.

PRESIDENT GETMAN — Dr. Pollack, do you want to answer that?

DR. POLLACK — I do not think that I can answer it. The two statements are in agreement. There just was no significant difference in the mortality ratio between the so-called labile and nonlabile groups. If the blood pressure fell very low, we did not separate it specifically in this particular study. Actually, we used the highest and lowest of each systolic and diastolic pressure, and averaged them together for the average pressure, but did not separate whether it was very low or very high.

PRESIDENT GETMAN — Dr. Harnes, would you like to comment?

DR. HARNES — I would agree with Dr. Pollack, the opinion being based on the results of our very extensive investigation of lability, using similar data to his. This included a study of lability from the medical history and we concluded that no consistent results were obtainable except in the one group of individuals whose blood pressure was observed to return to a level considered to be normal (below 140 mm. Hg systolic and 90 mm. Hg diastolic).

DR. BELL — Before concluding lability, we would like to say our data showed a mortality of 83 per cent in individuals whose blood pressure, after being initially high, dropped to completely normal limits, and this is very reassuring. But the question arises, suppose an individual on his initial examination has a blood pressure of 170/105 mm. Hg, and then on recheck it is 138/88? Should we completely ignore that, as our data would suggest?

These are certainly not the same sort of data presented some

years ago by Dr. Levy and his group, who studied a group of regular career Army officers who received annual examinations. They found that in those who initially had hypertension and then were allowed to rest until their blood pressure returned to a normal range, there was through the years a very definite increase in the incidence of persistent hypertension and its concomitant complications as compared to a completely normotensive group who did not present this hyperreactor phenomenon.

Insofar as our data are concerned, and although Dr. Harnes mentioned 83 per cent mortality, actually, the number of deaths in the entire group was only about 14. The statistical experts tell me that it is from the number of deaths in the group that the solidity of the conclusion is based, and that 14 deaths to them are not very many.

PRESIDENT GETMAN — Thank you, Dr. Bell.

You are all familiar with Dr. Kossmann and his work in electrocardiography. His fame is, indeed, international. His ability, both as a clinician and as a clinical investigator, is equally outstanding. He has been an active and productive member of the American Heart Association for many years, in spite of his youthful appearance. At the scientific meeting of the American Heart Association, he is acting as chairman for the session on instrumental methods and cardiovascular diagnosis.

It gives me a great deal of pleasure to introduce Dr. Charles Kossmann, Associate Professor of Medicine, New York University College of Medicine.

THE EFFECT OF MYOCARDIAL DISEASE AND DYSFUNCTION ON THE FORM OF THE ELECTROCARDIOGRAM

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Introduction

It is a special honor to address your Association on this occasion because it is, almost to the day, the twentieth anniversary of the first talk given to you by my former teacher, Dr. Frank N. Wilson. The paper he presented then,[1] along with another in 1942 which proved to be a classic,[2] covered almost all the new facts in electrocardiography which he and his associates had accumulated in the intervening five year period by means of the precordial electrocardiogram, utilizing the indifferent terminal of Wilson, Macleod and Barker.[3] In the fifteen years since 1942 surprisingly little of a diagnostically useful nature has been added to clinical electrocardiography which was not at least touched upon in that second paper.

So far as the electrocardiogram is concerned, the objectives of the medical department of an insurance carrier are obvious. In the broadest sense the insurance physician is trying to make an estimate of the probable duration of life and hence of insurable risk on the basis of an electrocardiogram. All physicians know the method is limited in this regard but in my experience only a few are aware of the considerable extent of these limitations.

As an illustration of opposite ends of the problem from the viewpoint of insurability, consider two patients. The first, a 53 year old physician, had begun to have pain of an anginal nature a few days before the first electrocardiogram was recorded (figure 1). It was not beyond normal in any respect. Four months later, myocardial infarction of moderately extensive degree occurred in the distribution of the anterior descending branch of the left coronary artery (figure 2). The second patient, a 61 year old

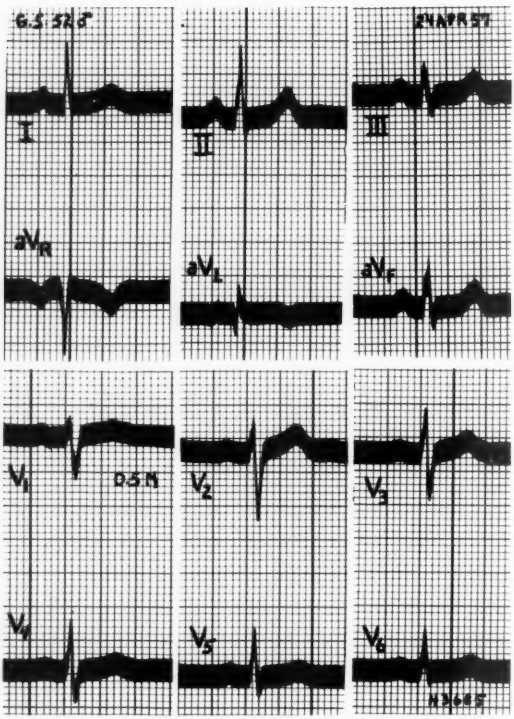

Fig. 1. Normal electrocardiogram of a white male, age 53, a few days after the onset of angina on effort. I, II, and III are the bipolar extremity leads; aV_R, aV_L, and aV_F are the augmented extremity potentials; and V_1 through V_6 are the usual precordial leads recorded at half normal sensitivity of the galvanometer (1 mv. = 0.5 cm.). Time lines occur every 0.04 sec.

woman, displayed a rather bizarre electrocardiogram which revealed no Q waves or S waves where expected, and displacements of the S-T segment and abnormal T waves in multiple leads (figure 3). Generally, such a record would be ascribed to coronary disease and recent myocardial necrosis. The disturbing aspect of the case was that an identical record was obtained ten years before (figure 4) and on numerous occasions between. Further,

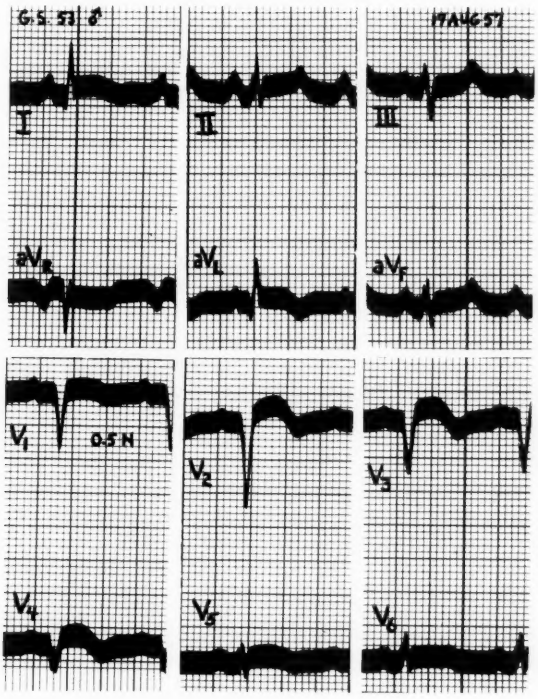

Fig. 2. Electrocardiograms of same patient shown in figure 1 recorded four months later after the occurrence of clinical myocardial infarction. Compared to figure 1 there are distinctive changes which accompany infarction of the anterior portion of the interventricular septum and the adjacent anterior wall of the left ventricle.

the heart size remained normal. There were no manifestations of diminished cardiac reserve, but the patient had a well fixed cardiac neurosis, iatrogenic in nature, as a result of having been hospitalized on several occasions for treatment of acute myocardial infarction which she never had.

The cases illustrate opposite ends of the insurance problem because, on the basis of the initial electrocardiogram alone, an insurance policy would have been issued to the first and refused

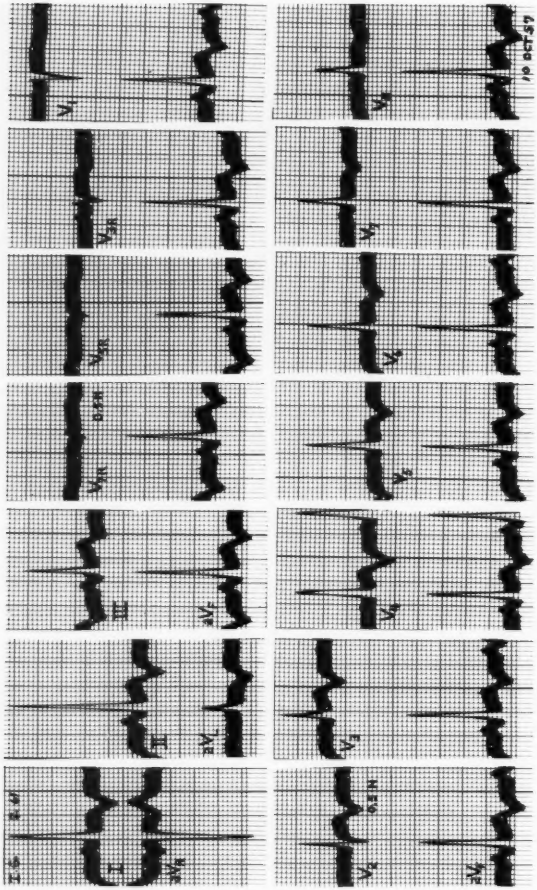

Fig. 3. Bizarre electrocardiograms of a 61 year old white female with no clinical evidence of heart disease recorded on October 10, 1957. Leads V_7 and V_8 are from the posterior axillary and midscapular lines at the same horizontal level as lead V_4. Leads V_{7R}, V_{5R} and V_{3R} are from the stations indicated by the arabic number but on the right side of the chest. Other symbols and calibrations as in figure 1.

to the second. In each instance these opposite decisions would undoubtedly have resulted in an eventual loss to the underwriter.

The key to the solution of such electrocardiographic dilemmas is, presumably, an objective of this presentation. Since my own major field of interest has been the clinical determination of heart disease rather than of insurability risk, my qualifications may not

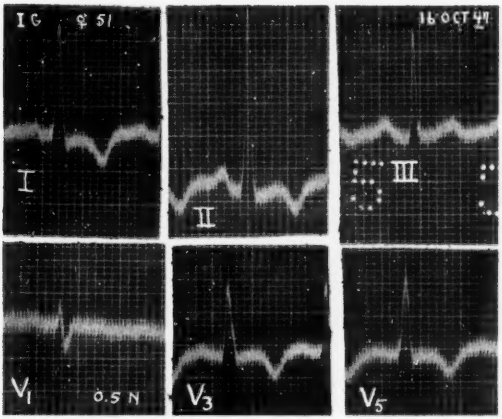

Fig. 4. Electrocardiograms from same patient as those shown in figure 3 but recorded ten years earlier (October 16, 1947).

be those required for such a task. Actually the prognostic aspect of a variety of electrocardiographic aberrations has been quite expertly handled in several publications by past or present members of your Association including, among others, Nyboer,[4] Bolt and Bell,[5] and Kirkland and his associates.[6] Further, it does not seem likely that the present electrocardiographic method, with one possible exception, can be refined to any further significant degree for purposes of determining insurable risk. Under the circumstances it would appear that an entirely new approach to your electrocardiographic problems must be considered in the not too far distant future. I am not at all certain what the nature of this approach should be. In the hope that I may just possibly ignite a blaze of ideas in this regard with some small sparks of information, I have elected to tell you something of the direction in which electrocardiography seems to be going and what changes may be made in the method in the future. If I seem, at times, too theoretical and insufficiently practical I hope you will bear with me. It is simply because we are both the victims of the circumstance that electrocardiography seems at the moment to be in a state of major transition.

Normal Electrocardiogram

Many of you will recall that in the first paper Wilson presented before you he discussed the interpretation of borderline electrocardiograms.[1] In it he made a plea for a more detailed study of the normal electrocardiogram by modern statistical methods, particularly those concerned with the determination of the skewness of the distributions and the predictability of the approximate frequency of any particular deflection. It was his impression at the time that insufficient data had been collected on the normal electrocardiogram and he expressed the hope that the medical departments of insurance companies would be in a position to fill this gap.

Since then many papers have been written on the normal electrocardiogram, particularly in children. The best of these and perhaps the most complete are those compiled by Ziegler[7] in children and by Simonson[8] in adults. On the other hand, most of these studies have not gone into greater statistical detail than to determine the minimum, the maximum, and the mean values of various deflections and intervals. It has not been possible to make accurate predictions of the frequency of variations from normal from these data as Wilson had hoped.

Several additional problems have arisen in the accumulation of information on the normal electrocardiogram since 1937. For example, the direct-writing electrocardiograph has come into fairly extensive use. Despite its great convenience for certain clinical emergencies, it has shortcomings which make for a variety of errors in the magnitude and form of electrocardiographic deflections. These errors are present in minute degree even when the instrument is in perfect working order; with hard wear and neglect these errors are usually magnified. Unfortunately, many of the studies of the normal electrocardiogram have been made with this relatively low fidelity instrument.

Other errors creep into the collection of normal data. One which is particularly disturbing is the contact of electrode jelly between contiguous precordial points. As those of you who do electrocardiography well know, such contact of the jelly will make for an area of leading from a very much larger portion of the chest than defined by the area of the electrode, and may account

for inversion of T waves in leads farther to the left than is actually the case. The precordial electrocardiogram thus obtained will be in error. It is difficult to prevent even good technical aids from making this mistake occasionally.

Several years ago I made an attempt to put together all of the available normal series into some kind of a usable group of tables.[9] Actually, four separate tables were designed to cover the age groups in years from 0 to 1, 1 to 10, 10 to 20, and above 20. Regrettably, all that could be included in these tables were the maxima, the minima and the corrected means, depending upon the number of variants which each author provided in his series. Clearly such tables are clinically useful but are probably inadequately refined for the rigid requirements of the insurance physician.

It would seem that although two decades have passed since Wilson pointed out the need for more adequate normal data, these data have not been accumulated except in a less than ideal manner and without detailed statistical treatment. It is not likely that a statistically mature and comprehensive study of the normal electrocardiogram will be made by a clinician because he usually has access in any one patient to other data—historical, physical, laboratory—which soften his requirements for a precise definition of the normal electrocardiogram. Further, he is all too often confronted with the type of problem shown in figures 1 and 3, where the apparent absence of correlation between the anatomic and the electrophysiologic was misleading diagnostically.

It would still seem, as Wilson indicated in his talk twenty years ago, that the undertaking of a careful statistical study of the normal electrocardiogram would be worth while, particularly to the insurance underwriters of America who stand to benefit most from a sharp definition of the dividing line between the normal and the abnormal. Particularly valuable in this regard would be additional observations on the normal electrocardiogram in adults between the ages of 40 and 60. This is the one area, mentioned earlier, in which it is believed that present day electrocardiography can be improved from the insurance physician's point of view.

Electrocardiographic Leads

In the last decade or so, much information has been accumulated on the parameters which determine the form of the normal electrocardiogram. These are: the source of potential, the external medium, the boundary of the external medium, and the internal medium (figure 5). When dealing with the heart as a whole,

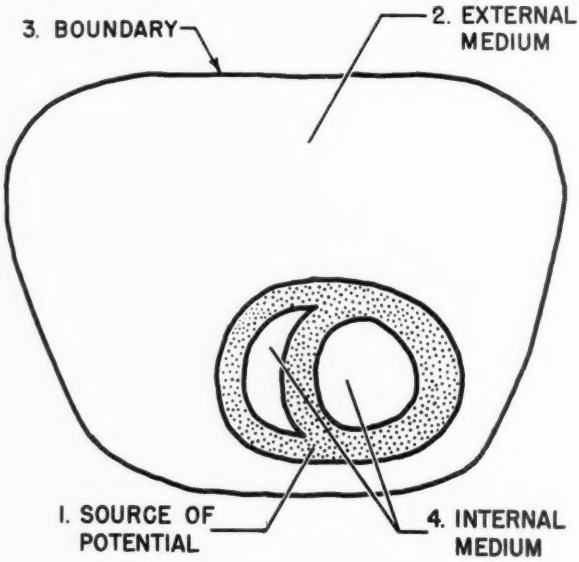

Fig. 5. Cross sectional diagram of the thorax showing the four basic determinants of the form of the electrocardiogram. The source of potential (1) is the myocardium, and the form of the electrocardiogram it determines will depend on whether it is atrial, ventricular, or pacemaker tissue; the external medium (2) is composed of the body tissues; the boundary (3) is the body surface; the internal medium (4) is the blood in the heart.

other factors such as intra-atrial and intraventricular conduction play an important part in determining the contribution by the source of potential to the form of the electrocardiogram.

The source of potential is the myocardial cell. Actually, a

voltage difference exists across the membrane of the cell and is spoken of as the transmembrane potential. We, and others before us, [10, 11, 12], have been able to measure this potential by puncturing the cell membrane with a special recording microneedle. The record which is obtained is known as the monophasic action potential. Its form was determined many years ago by the physiologists who obtained it not by the intracellular technic but by placing one electrode of a bipolar lead on an injured end of the cell (injury potential).

The monophasic action potential is converted into a multiphasic record by virtue of the fact that it is surrounded by a conducting medium.[13] Until 1947 it was customary to regard this conducting medium as relatively infinite and homogeneous, the former of which is obviously quite far from the truth. Actually, the surface of the body acts as a boundary of the medium. Since the medium is irregular and bounded the electrical field resulting from a source of potential within it is distorted. The questions that have received much attention in the past decade are what is the degree of this distortion and how can it be measured.

Burger and van Milaan[14] first demonstrated how it could be measured and this resulted in the introduction of the concept of the "lead vector". We have for years spoken of the heart vector; perhaps you know it better by the term "electrical axis". The lead vector, on the other hand, was new with the writings of Burger and van Milaan, although the principle involved was not new and had been used in a variety of scientific fields for a long time.

Actually, what Burger and van Milaan,[14] and later Frank,[15] were trying to do was to free clinical electrocardiography from some of the restrictive assumptions which were included in Einthoven's hypothesis. They felt that three assumptions, namely that the body is a large conducting medium, that the medium is homogeneous and resistive, and that the heart is at the center of the medium, could be eliminated from the realm of electrocardiographic theory. The other restrictive assumptions, namely that the source of potential may be treated as an equivalent dipole and that the dipole undergoes no change in position during the cardiac cycle, could not be handled in the manner to be indicated.

Perhaps the concept of the lead vector can best be approached by way of the equilateral triangle with the heart or source of potential regarded as being at its center (figure 6). Using Einthoven's formulations and notations, in this case in lead I, it is clear in the figure that e_1, the projection of E (the heart vector) on lead RL, has the value $e_1 = E \cos \alpha$, and that when E is horizontal $e_1 = E$ and when E is vertical $e_1 = 0$. These relationships, of course, are made use of everyday in clinical interpretation of a record when determining the direction of the electrical axis.

Of significance is that the assumption of a symmetrical field and equidistant lead points (equal sides of the triangle) make

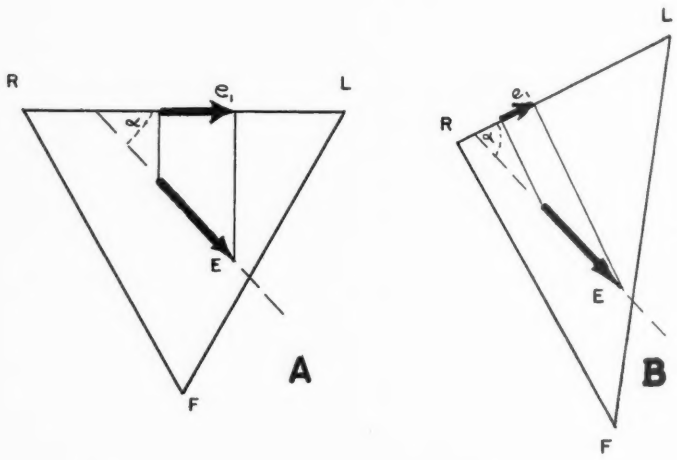

Fig. 6. Diagram of an Einthoven triangle (A) which is regarded as geometrically and electrically equilateral, and a Burger triangle (B) which is an electrical representation of the inequality of contributions of the bipolar leads by virtue of geometric inequality of the distance of the heart's center (heart vector, E) from the apices R, L, F. The Burger triangle really is the inverse of the geometric situation — a short distance between the heart center and the lead point results in a long length (large scalar magnitude) of the lead. The diagram attempts to emphasize the determination of the voltage of a lead not only by the magnitude and direction (angle α) of the heart vector with respect to the lead, but the relative length (electrical) of the lead itself (lead vector).

it unnecessary to use any correction for the leads which is required in a distorted field. The leads RL, RF and LF (I, II, III), being equal in length, are affected by the heart vector similarly and, hence, are regarded as unity in any calculations that are made. Thus, expressions for e_1, e_2 and e_3 which are the projections of E on the sides of the triangle are only in terms of the heart vector E and the effect its angulation with the respective lead has on the recorded potential.

Let us suppose, on the other hand, that the source of current is eccentric with respect to points R, L and F so that it is closer to L than to R. Under these new circumstances the scalar magnitude of lead LF will be larger than of RF. This difference can be represented geometrically in a reverse way by lengthening lead LF in the triangle which then becomes scalene in form. It can be seen at a glance that the projection of E on the leads will not be the same as in the equilateral triangle, not only by virtue of the different angulations between the two but also because the different rather than equal distances between lead points result in different "weights" proportional to the different lengths of the three leads. Each lead, having direction and length, meets the requirements of a vectorial quantity — the lead vector. It also bears other names such as "image vector", "coefficient of correction", "transfer function", and "transfer impedance".

It is obvious that in leading from such an asymmetrical field the factors of length and direction of the lead cannot be ignored. The original simple expression for the projection on lead I now becomes $e_1 = \overline{RL} \cdot \overline{E} \cos \alpha$. In this new expression, then, the scalar quantity obtained in a lead is really the product of a vector and the projection of another vector on it. These two vectors are called respectively the "heart vector" and the "lead vector".

Clearly, if the principles above apply to bipolar leads, they also apply to unipolar leads which are really bipolar leads with one pole at zero potential.

Looking at the last expression it can be seen that there are three unknowns. The lead e_1 is easily obtained but the problem that has always faced the electrocardiographer is how to estimate

the relative importance of the heart vector and the lead vector in the determination of the form and magnitude of the lead. Stated clinically, the physician wants to distinguish between the electrocardiographic effects of intrinsic cardiac disease or dysfunction, and the effects of an unusual position of the heart in the chest or an unusual distortion of the chest itself.

Once the lead vectors (correction coefficients) are determined they can be used to obtain the correct "components" of the heart vector. Since the latter is a spatial entity it is mathematically expedient to break it down into its horizontal, vertical, and sagittal components (x, y, z). If these orthogonal components can be corrected with the necessary coefficients electrocardiograms or vectorcardiograms can be recorded undistorted by the effects of an irregular body surface, inhomogeneities, or an eccentric source of the current.

Unfortunately, this cannot be done directly with any degree of accuracy in living man, although we have developed a technic which shows promise.[16] It can and has been done on models by Burger and van Milaan,[14] by Frank[15] and by Schmitt and Simonson.[17] The only potential shortcoming of these investigations is that they have been done on models. It is not known whether the various lead systems which have been designed recently on the basis of these experiments, and there are a good number of them, do in truth yield the rectilinear components of the heart vector in all subjects with all types of heart disease. Clearly, a very large heart yields an equivalent dipole which has a different location than one which is small. Voluminous lungs may also change the heart center location. It is not known whether a lead system which is proven to give the correct rectilinear components in the normal subject will also yield these components of the heart vector in the abnormal subject.

If the lead vectors for the bipolar extremity leads are determined as described above or in other ways, either in models or in man, and arranged as a triangle, the latter is called the Burger triangle. It may be regarded as a generalization of the Einthoven equilateral triangle. It usually has an asymmetrical scalene appearance with lead III larger than lead II, but this

varies depending on the location of the heart or dipole center assumed by the investigator.

Implicit in calculations made either with the Einthoven or Burger triangles is that the source of potential in the heart can be regarded as an equivalent fixed position dipole. There is evidence both for and against the dipole assumption which will not be presented at this time. It should be noted too that in the Burger concept the position of the dipole is assumed to be fixed. It is almost certain now that in a large heart, and particularly when there is block of the impulse to one or the other ventricles, the position of the dipole does in truth vary. This makes the problem much more difficult to solve exactly.

McFee and Johnston[18] have pointed out that the lead vector concept requires the retention of one of Einthoven's postulates, the validity of which is questioned by many. This is the assumption that the electromotive force of the heart may be regarded as an equivalent dipole. To overcome this restriction they have developed the concept of the "lead field" which may be regarded as a generalization of the lead vector concept. A discussion of this interesting point of view is beyond the bounds of this paper.

From the foregoing it must be obvious that not a great deal of a definitive nature can be said of the innumerable accessory or supplementary leads which have been designed in great numbers in the past. In a comprehensive review of all the published data on the subject Berger[19] has come to the conclusion that additional leads are rarely diagnostic but often confirmatory of abnormalities already present in more subtle form in the usually recorded extremity and precordial leads. The problem revolves to some extent around the question of whether the heart behaves as an equivalent dipole of variable moment. If it does, then only three properly factored bipolar leads from four surface points should be necessary to obtain all the desired, three dimensional electrocardiographic data. If on the other hand it is believed that a near exploring electrode will yield "local effects" of the heart, and most recent data favor this view, then some exploration of the precordium in the present or some other manner will continue to be necessary for a complete survey.

In this regard a lead from high on the right precordium at the approximate level of the second rib at the left sternal edge is regarded by many as an essential area to explore to reveal particularly the late positive QRS deflection ascribed to excitation of basal portions of the heart.

Spatial Vector Electrocardiography

The above considerations of the heart vector make it obvious that information obtained by synthesis of a single vector from the usually recorded scalar leads can only be a first order approximation of the truth, however valuable it may be otherwise. It is for this reason that the perpendicular plane method of determining the spatial vector, such as was designed by Grant and Estes,[20] can serve only a limited purpose in insurance medicine. As a matter of fact, for purposes of analysis a mean vector is less desirable than the scalar leads, particularly since the diagnostically important deflections at the beginning and at the end of QRS (the Q wave and the S wave) are obscured in the mean vector electrocardiographic method. There was hope for a time that the angle between the mean manifest QRS and T vectors estimated by this method would have clinical usefulness. This angle can be calculated mathematically with the aid of tables or by means of a variety of devices. Although the maximum normal value originally given was 50°, later observations indicate that the 95 per cent range is probably in the neighborhood of 7° to 90° in middle aged men and is even greater in overweight subjects.[21]

In conclusion it may be said that vector electrocardiography as a clinical method is interesting and useful but being a generalization of the scalar method, tends to obscure some of the details of clinical value revealed by the latter method. Whether the application of the lead vector concept to the method will increase its precision can only be decided in the future. Interestingly enough one study thus far has demonstrated surprisingly little effect of an orthogonalized axis system on the variability of the QRS and T vectors and the angle between them when compared with the results of an equilateral tetrahedral reference system.[22]

Vectorcardiography

Most recent progress in vectorcardiography has been centered around the design of an orthogonalized system of three dimensional leading which will yield as accurately as possible the true rectilinear components of the electrical record free of the distortion caused by irregularity of the body surface, inhomogeneity of the surrounding medium, and eccentricity of the heart. To restate this and relate it to what was said above, the problem has been to determine the lead vectors (or lead fields) for some system of leads which will yield the horizontal, vertical, and sagittal components of the heart vector. Only when these are available can a true vectorcardiographic trace be obtained.

Somewhat disturbing is the fact that the various systems of obtaining vectorcardiographic leads have not received an adequate comparison with our present methods of leading which utilize the Einthoven triangle and the chest leads. Until this is done, I do not believe that any one system can be adopted for general use, or indeed that vectorcardiography can be adopted at all in any form for clinical purposes.

Judging from our half century of experience with the Einthoven triangle, it is very probable that some semiorthogonal system will eventually be adopted for routine clinical use even though it may not meet the rigid requirements of the engineer or the physicist in every case. Ideally, the lead vectors should be determined in each patient individually but the probability of such a procedure being practical is small, if it is ever actually provided. The so-called quantitation of electrocardiography, therefore, is approaching a second order of magnitude but the approach will require compromises with practicality if the method is to continue to maintain its valuable place in clinical cardiology. This probably applies with even greater emphasis to insurance medicine.

Remarks on Current Concepts of the Spread of Excitation and Recovery in the Walls of the Ventricles

As a result of recent experiments in several laboratories, the

details of Lewis' concept[23] of the spread of excitation through the ventricular walls have been challenged. The principal points of difference concern the ventricles and may be stated as follows: (1) The midportion of the left side of the interventricular septum rather than the base is excited initially and a considerable portion of it excited from below upwards; (2) a variable proportion of the subendocardial myocardium is excited simultaneously and the remainder more slowly, rather than in a uniform endoepicardial manner; (3) there is a physiological barrier between portions of the septum excited from either side which becomes manifest when unilateral block is produced. Since there is at present no universal agreement on the validity of these challenges, especially the second, and since the details of the experimental data from different sources vary, a review of the present status of the intramyocardial spread of excitation in the ventricles is in order.

Figure 7 shows diagrammatically one type of spread of excitation in a normal subject determined from body surface and endocardial leads. It is obvious now, principally from the work done by Bryant,[24] that there are differences in the sequence of excitation of the two ventricles in different normal subjects; and these may vary over the spectrum of complete excitation of the septum from left to right to what is usually regarded as complete left bundle-branch block. His interesting observations explain in part the chaos which exists with regard to the interpretation of records presumed to indicate block of the right bundle-branch, either incomplete or complete, and of records which indicate hypertrophy of the right ventricle. They also emphasize the very considerable importance of making electrocardiographic records at faster than the usual paper speed, and of taking certain chest leads at high gain and at high levels on the right precordium. His data, to be published soon, may have considerable interest to insurance medicine.

Because most of the older data on the spread of excitation through the walls of the ventricles are based on measurements made at the endocardial and epicardial boundaries, groups of investigators working in the laboratories of Prinzmetal et al,[25] Scher and Young,[26] Durrer et al[27] and Sodi-Pallares et al[28]

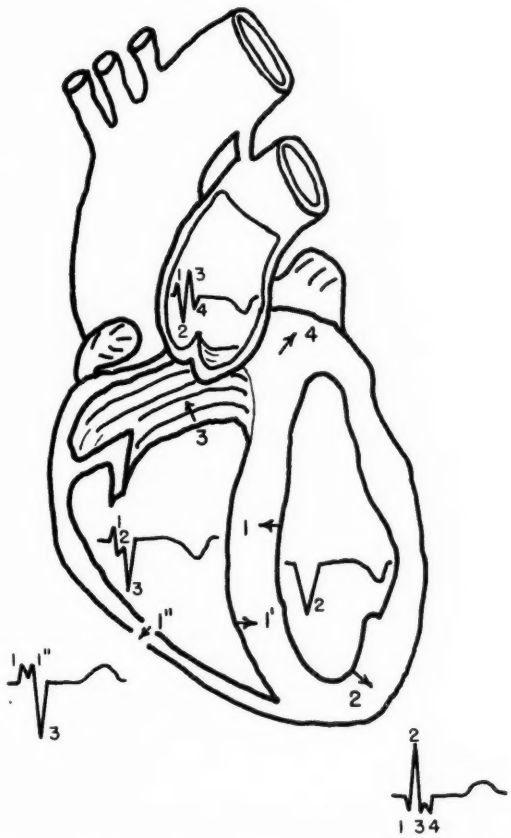

Fig. 7. Diagram of one type of spread of excitation in the normal heart. Numbered vectors in the heart correspond to numbered deflections in the various intracardiac and external leads. The separation of deflections has been exaggerated for clarity.

have undertaken a study of the electrical potentials within the left ventricular wall (intramural potentials). All have been made with some variation of a stab or plunge electrode. Both unipolar and differential records have been made and the experimental

animal has usually been the dog but some observations have been made in man.

Unipolar records have disclosed an absence of an initial R wave in leads from a variable portion of the wall under the endocardium. Differential records have revealed instantaneous excitation and sometimes reversed excitation (epi-endocardial) in a similar part of the wall. The laboratories, the methods of leading and the percentage of subendocardial wall thought to be instantaneously excited are as follows:

Investigator	Type of Leads	Per cent of Subendocardium Excited Instantaneously
Prinzmetal et al	Unipolar	80
Sodi-Pallares et al	Unipolar	66⅔
Durrer et al	Unipolar, transmural, partial transmural and multiple differential	40
Scher et al	Multiple differential	close to 0

Later experiments by the first two groups listed have reduced their original estimates somewhat. In general the results have been interpreted to mean that Purkinje's fibers extend out into the wall although never demonstrated anatomically in the human heart. They have also been extended somewhat prematurely to the clinical interpretation that only the outer portions of the ventricular wall contribute to the R wave in an epicardial or semidirect lead and, therefore, subendocardial infarction can occur without electrocardiographic manifestations. There appears to be much clinicopathologic evidence in infarction to refute this interpretation.[29, 30] The fact that an occasional heart may be found at necropsy with subendocardial infarction but with no electrocardiographic abnormalities is no argument at all unless frequent records were made from the time of onset of symptoms until death and unless a preinfarction record was available. The readiness with which an almost monophasic record can be produced by pressure of an intracardiac electrode on the endocardium leaves no doubt about the normal electrical behavior of subendocardial muscle varying in no way from other myocardium. Any difference must be ascribed to factors other than the intrinsic electrical properties of the involved cellular membranes.

It is our considered opinion that the variability of experimental results with intramural leads does not permit any definitive conclusion regarding their significance, and that the interpretation that subendocardial infarction is electrocardiographically silent is probably in error.

An Insurance Aspect of Electrolytes and the Electrocardiogram

It is not likely that an electrocardiogram showing the striking abnormalities from electrolytic and acid-base imbalance, summarized in figures 8, 9 and 10, will ordinarily be encountered in

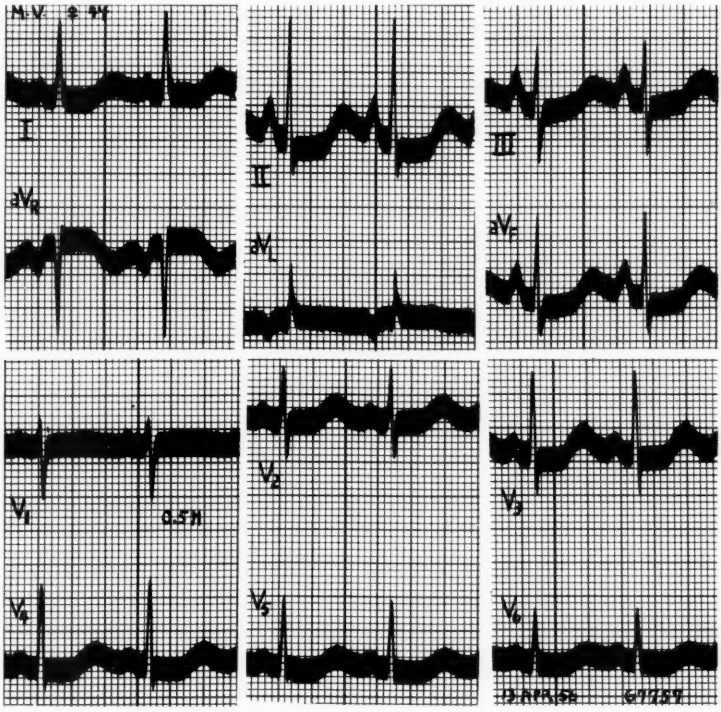

Fig. 8. Electrocardiogram of a 44 year old white female (M.V.). The patient had duodenal obstruction and had been vomiting for the previous three weeks. The modifications in electrolytes present at the time of the electrocardiogram are shown in the table of figure 9.

PT. M. V. ♀ 44	13APR56	15APR56	17APR56	18APR56
EKG	ABNORMAL	——	NORMAL	——
BUN	37.1	21.4	——	23.7
NA^{+}	138.0	135.0	——	135.0
K^{+}	2.0	2.8	——	4.8
CL^{-}	——	86.9	——	97.6
HCO_3^{-}	52.2	34.0	——	27.3
CA^{++}	11.1	——	——	——
P^{+++}	3.8	——	——	——

Fig. 9. Table showing the time of recording the electrocardiograms reproduced in figures 8 and 10 in patient M.V., in relation to the serum electrolytes and blood urea nitrogen before (April 13, 1956) and after the institution of medical therapy to correct the imbalance.

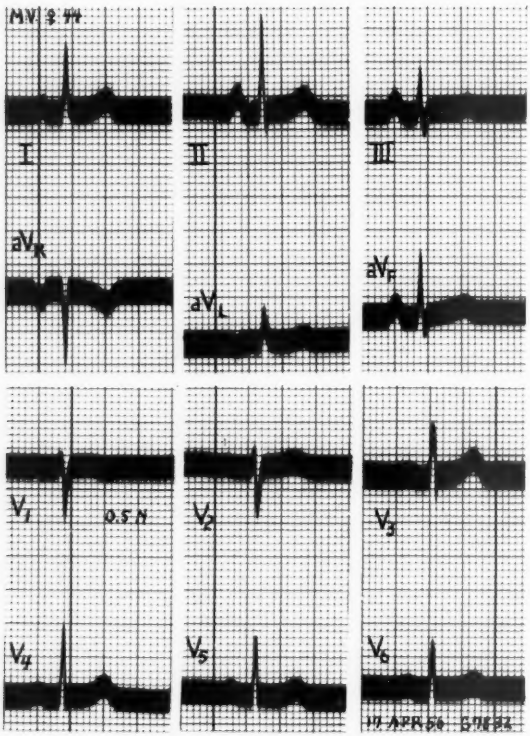

Fig. 10. Electrocardiogram of patient M.V. recorded four days after the one shown in figure 8, and after the correction of the hypochloremic alkalosis with azotemia and hypokalemia present at the time of recording the latter (figure 9).

insurance medicine. It is shown simply to emphasize how radically such disturbances, in this case caused by persistent vomiting secondary to duodenal obstruction, can modify the record. More likely to be encountered is the electrocardiogram on the right of figure 11. This 51 year old man presented himself initially with some pendant edema and an electrocardiogram with minor abnormalities of T wave as shown on the right. The physician in charge regarded him as having coronary disease and heart failure.

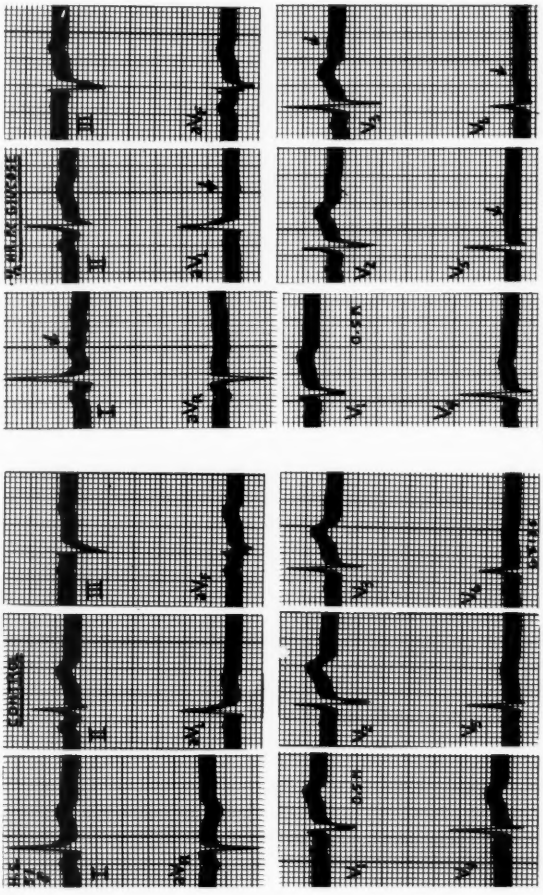

Fig. 11. Electrocardiograms of a 51 year old white male before (left) and one half hour after the oral ingestion of 100 grams of glucose in water. Arrows in the latter show the changes in T wave and U wave, particularly in leads I and aV_L, which were caused presumably by the reduction in serum potassium caused by the temporary alimentary hyperglycemia. The man displayed no other evidence of heart disease.

The figure illustrates the interpretive difficulties which may arise if electrocardiograms are not made in a basal state.

Another electrocardiogram (left of figure 11) was recorded with the patient in a basal state which was normal. Then 100 grams of glucose were given in water by mouth. One half hour later the electrocardiogram displayed minor but distinctive abnormalities of the T wave which I am sure would give concern to any examining physician. With this information a clinical review of the situation was made. There was no other evidence of heart disease, and the edema was found to be on the basis of an old deep vein thrombophlebitis.

The changes induced by the glucose are usually ascribed to a reduction of serum potassium, although it was not measured in this instance. The example points up the need for recording electrocardiograms for insurance purposes with the applicant in a basal state, and the value of this simple test for resolving similar problems.

Stress and the Electrocardiogram

In the time remaining I will not attempt to give you a comprehensive survey of the present status of the exercise tolerance test in clinical medicine. I will instead give you some opinions based on a personal clinical experience on those aspects of the test which have impressed me the most.

From electrocardiograms which have been sent to me for opinion at various times it is quite apparent that most physicians do not know how to do an exercise tolerance test. Usually, what is submitted are twelve control leads and twelve others labelled "after exercise". Obviously, with a single-channel instrument the twelve postexercise leads were recorded over a period of time which might have been anywhere from two to ten minutes, during which time the effects of exercise, if any, would have disappeared. It is best to record as few leads as possible if a single-channel instrument is available. Leads I, II, III, and V_5 should be the maximum.

Often it is obvious that a sufficient amount of exercise has not been given because the pulse rate is changed slightly if at all.

In interpretation, care must be exercised to use the P-R segment as the reference level. It often is displayed downward after

exercise giving the false impression of a downward displacement of the following S-T junction (figure 12).

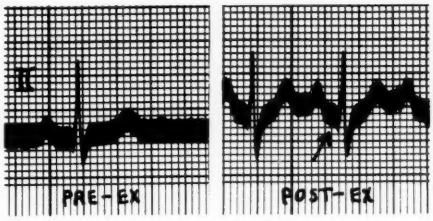

Fig. 12. Depression of the P-R segment in lead II (arrow) caused by exercise in a normal subject giving the false impression of depression of the S-T junction. Pre-ex, before exercise; post-ex, after exercise.

Lastly, an exercise tolerance test properly done is an exceedingly valuable diagnostic aid. We obtain a very considerable percentage of positive results, as shown in figures 13 and 14. The reason for our success is probably due to the fact that the test is most often done on the large number of patients who have a history of distinctive anginal pain but in whom the resting electrocardiogram is normal. It is the exceptional case in which the test is done because a decision cannot be made clinically regarding the origin of the pain. Oddly enough the results in these are usually negative or equivocal. In applicants for insurance it is likely that the percentage of positive tests will be much lower than indicated above, even when the test is performed satisfactorily.

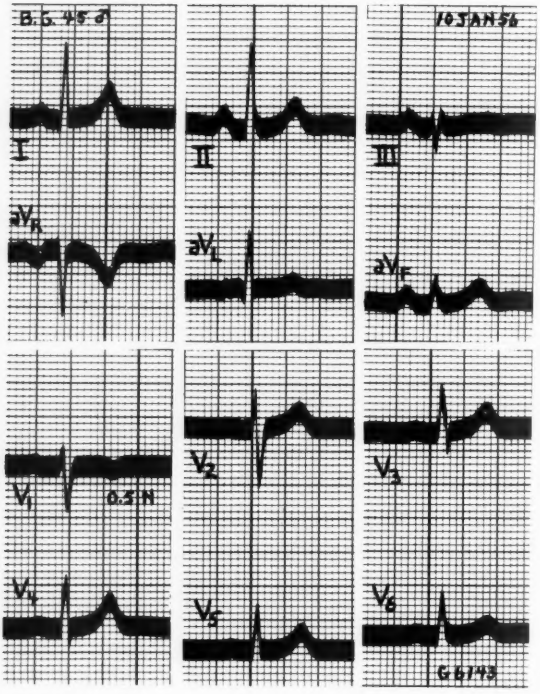

Fig. 13. Normal electrocardiogram of a 45 year old white male (B.G.) who had typical angina on effort for the previous nine months.

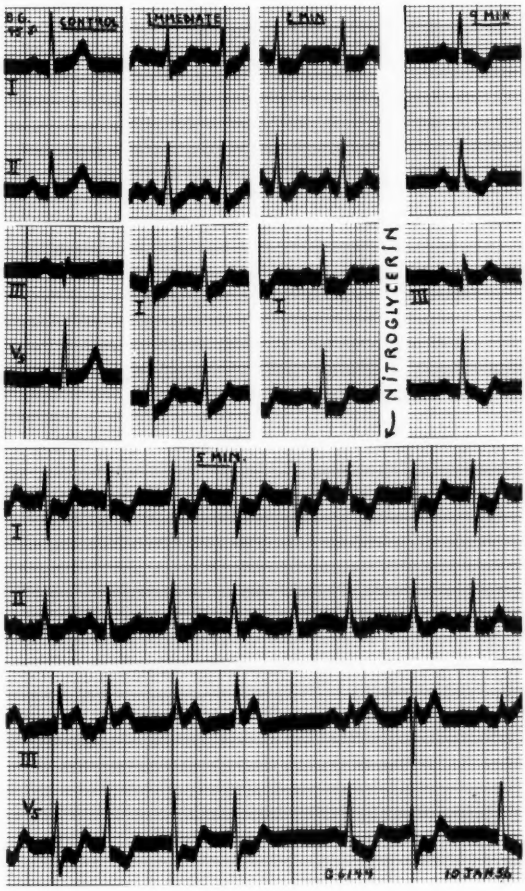

Fig. 14. Leads I, II, III, and V_5 made before and after exercise of the same subject (B.G.) on the same date as the electrocardiogram shown in figure 13. The exercise consisted of stepping up and down on a stool 12 inches high, 45 times in 1½ minutes. Retrosternal pain was produced, and after the 2 minute recording nitroglycerin was given. At 5 minutes, clusters of ectopic ventricular beats occurred intermittently. At 9 minutes the T waves were still strikingly abnormal. Two years later this patient's resting electrocardiogram was still normal.

In the recordings immediately after and 2 minutes after exercise, lead I was recorded instead of lead III by mistake.

Summary

In appraising the effects of some diseases and dysfunctions on the electrocardiogram the conclusion is reached that electrocardiography in its present form does not have much more to offer in determining insurability risk than it did a decade or more ago. A more meticulous study of the normal electrocardiogram could conceivably increase the usefulness of the method in medical underwriting problems but this is not likely to be great. However, fundamental advances are being made especially in relation to the effect of asymmetry of the electrical field of the heart on the magnitude and form of leads made from the surface of the body. These advances bode well for the eventual adoption of a more quantitative system of leading which may have certain diagnostic advantages over the leads now in use. The importance of properly evaluating the effects of physical and chemical stress on the electrocardiogram for determining the presence or absence of organic disease is reiterated.

PRESIDENT GETMAN — Thank you very much, indeed, Dr. Kossmann. I have asked two friends of ours to discuss Dr. Kossmann's paper informally — Dr. Paul Langner, and Dr. Harry Kirkland, whom you all know very well.

DR. HENRY B. KIRKLAND — This very erudite paper has, obviously, material for Paul Langner to discuss rather than myself. I am simply going to confine myself to one or two little remarks.

Over the years, we have given a lot of thought to the possibility of running an extensive series of normal electrocardiograms. Whether we are right or wrong in our decision, I do not know, but it has been felt that the practical gain to us would probably not be worth the very great trouble that it would require.

The second thing on which I should like to comment is this matter of electrolyte balance. It is unquestionably a very troublesome and confusing thing, and I am sure you will agree that we have all been sorely put to it on occasion when we are dealing with an important case, particularly an over age applicant. We are confronted with an abnormal electrocardiographic pattern, which is indistinguishable, as far as the actual background is

concerned. We do not know whether it is due to coronary insufficiency or to electrolytic balance, and consequently, must be very conservative in our attitude.

The last remark I should like to make is about the stress test. It is extremely gratifying to hear Dr. Kossmann point out, as he has done so well, that we frequently encounter exercise electrocardiograms which are completely inadequate from a technical standpoint, and we certainly want to be very careful in evaluating them.

DR. PAUL H. LANGNER, JR. — Dr. Kossmann has certainly given us some very provocative material to consider. I believe that if we are going to make further progress in electrocardiography, we do need a new approach. However, please be reassured that it will be some time before this new approach and the new methods can conceivably supplant the present-day practices with which you are all familiar, so, if one or two of the points that he made were not readily understood it is no cause for concern.

I have been asked to say a few words about the present status of vectorcardiography in relation to the insurance business. Until recently, most vectorcardiograms have been made from the cube and the tetrahedronal reference frames. In view of recent developments, which Dr. Kossmann has so beautifully described, it would seem that these older systems appear to be totally inadequate, except for very rough approximations. They introduce distortion, because they are not based on sound physical principles.

For instance, the use of anatomic distance to determine electrode placement, such as they do in both the cube and the tetrahedron, results in significant error. In vectorcardiography, as well as in electrocardiography, we must think not in anatomic distances, but in terms of electrical field distances which are quite different, as Dr. Kossmann has emphasized. In other words, when he was talking about lead vectors and image surfaces and eccentric dipoles, he was giving you the quantitative basis, the stuff of which orthogonal and proper lead systems are made.

Furthermore, we must make provision for the eccentricities

of the heart bipole, the variations in the bipole location, and even the possibility that there may be multiple dipoles scattered throughout the heart area.

Appreciation of these factors indicates clearly, I believe, that the cube and the tetrahedronal methods of vectorcardiography are outmoded.

Before we can use the vectorcardiogram with a reasonable degree of effectiveness and obtain some semblance of quantitative results, we must employ systems which compensate for these factors which I have just mentioned, and which Dr. Kossmann pointed out. Methods to accomplish this have been devised, and an excellent example is the system of Otto Schmidt, which has been devised in a three-dimensional torso model. Its limits of accuracy have been determined.

There is promise that vectorcardiography will eventually prove to be useful in life insurance medicine, but only after we have accumulated and studied the data, improved with these new more quantitative systems.

We have, as Dr. Kossmann may know, recently compared four of the orthogonal systems in the same series of patients, and the results were practically interchangeable in a large majority of the cases. Now, it is a small series — there were about 60. But it is, I hope, a step in the right direction.

In addition to having a good reference system, that is, the electrode placement on the body, one must have a suitable way of displaying the loop itself. There are three ways of doing this. So far, the most common method has been to use the three projections, that is, the frontal plane, the horizontal or the plane of the chest leads, and the sagittal — cutting them through the middle. This method leaves much to be desired.

The second method is the actual three-dimensional stereoscopic visualization of the loop, just like the old-fashioned stereopticon you used to use in the parlor. This is very interesting, but is principally a visual impression that must be dealt with in descriptive terms, and does not lend itself readily to quantitative study.

There is a third method — I am not giving you all the methods,

but just three — and this, I believe, is the most promising. It is obtained by use of what we call a revolver. It is a rather electronic device which permits one to rotate the loop and look at it from any angle.

Using this revolver and some of the others, we have found that the normal loop is approximately in a plane, that is, the whole loop lies in a plane surface. By means of the revolver, you can get the broad side or open view of the loop, and it provides a relatively simple means of showing the complete vectorcardiogram in a single picture. In essence, it reduces the three projections that you are used to seeing to a single picture, plus two measured angles which give the orientation of the loop in space.

This method has the advantage of separating the orientation of the loop in space from the actual shape of the loop, and the ability to study these two items separately, I think, should be of great advantage.

In conclusion, there will certainly be things apparent to you in this type of vectorcardiography that cannot possibly be seen in the scalar leads, as usually recorded; and I believe that we will soon have useful information which could not possibly be obtained in any other way. So, vectorcardiography is coming into its own. However, there are two reasons why I do not believe that it is going to be of any immediate help to us in life insurance medicine.

The first is the obvious one — the lack of available equipment, except in research centers, and I mean good equipment.

The second is the fact that we need to do much more work in accumulating and analyzing data obtained from these more nearly orthogonal systems.

PRESIDENT GETMAN — Thank you, Dr. Kirkland and Dr. Langner. Are there any questions that you would like to ask Dr. Kossmann?

I think that we all have been looking forward to hearing the next speaker with a great deal of interest. His reputation in the field of cardiac surgery is now worldwide. His technical genius is matched only by his ingenuity in the development of newer and more remarkable surgical procedures.

We are most fortunate, indeed, to have Dr. Charles P. Bailey, Professor of Thoracic Surgery of the Hahnemann Medical College and Hospital of Philadelphia, with us today. We are particularly gratified that he could find time from his busy schedule to come here. The title of his paper is "Recent Advances in Surgical Correction of Congenital Heart Lesions", and, perhaps, we may get him to say something about acquired heart disease.

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RECENT ADVANCES IN SURGICAL CORRECTION OF CONGENITAL HEART LESIONS

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Our discussion will be focused on congenital heart disease, but a couple of facets of that old worn-out subject of mitral stenosis might be of interest. Recently, there have been some new and stimulating developments.

Congenital heart disease, of course, is something that one is born with. It cripples in a purely mechanical fashion. Initially, the myocardium is excellent, and it may preserve pretty good health for many years. The essential problem is that the myocardium is overworked because of the inefficiency engendered by the mechanical abnormality. Therefore, one logically may expect that if such a patient is operated on in time, and if the abnormality is corrected totally, he will be restored to normality, and will be able to live a life of normal activity and duration. Fortunately, that hope often is not too far out of line.

Cardiac catheterization, particularly right-sided cardiac catheterization, is probably our most important diagnostic measure in congenital heart disease. Most of these deformities affect the right side of the heart, unlike the acquired lesions which usually affect the left. Hence, right-heart catheterization is an eminently satisfactory investigative technic.

We must not overlook the value of angiocardiography; but, as time goes on, it seems that the catheterization expert is becoming more and more able to diagnose obscure heart disease. Hence, the call for angiocardiography is less frequent.

Patent ductus arteriosus is commonly thought of as a congenital lesion. Of course, it is present at birth and we usually date the advent of elective heart surgery from Gross's[1] first successful case of ligation of the patent ductus arteriosus in 1939. However,

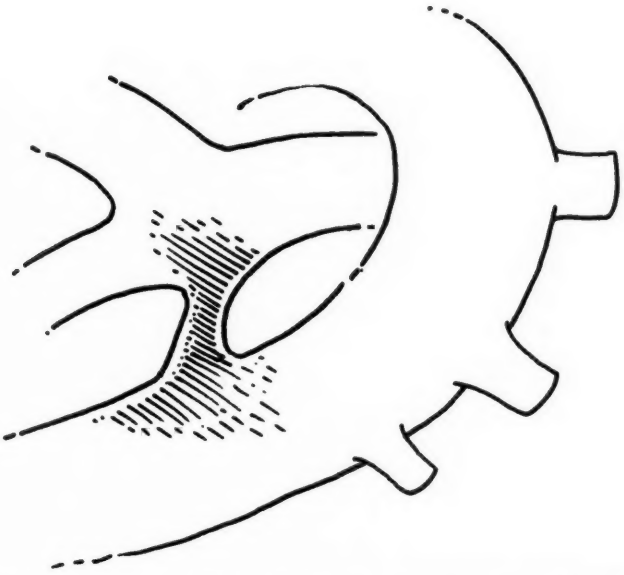

Fig. 1. Diagram of a patent ductus (the shaded portion), forming a channel for blood to shunt from the aorta into the pulmonary artery.

strictly speaking, a patent ductus arteriosus is not a congenital defect. It is rather a developmental abnormality due to failure of proper involution of a normal fetal structure (figure 1).

Its function, prior to birth, is to by-pass the collapsed lungs which present great resistance to blood flow. The right ventricular output readily passes through the ductus into the aorta, and so nourishes the body without going through the functionless lungs. Within a few moments of birth, apparently synchronous with the first inflation of the lungs, the pulmonary vascular resistance falls, rendering it easier for the blood in the pulmonary artery to traverse the lungs than to be shunted into the aorta.

It has been suggested that sometimes there may be less than the normal amount of muscular tissue in the wall of the ductus. Hence, complete obliteration of the passage cannot take place

following the opening up of the pulmonary vascular bed. One per cent of individuals do have a patent ductus arteriosus. Otherwise, these persons are entirely normal.

As the infant develops, the aortic pressure rises to adult level. This permits a left to right flow of blood from the high pressure aorta into the relatively low-tensioned pulmonary artery. This blood which has been oxygenated, having passed through the lungs and returned to the left side of the heart, must traverse the pulmonary capillary bed once again. This causes engorgement of the pulmonary vascular bed.

One point should be stressed before we discuss cardiac septal defects. A patent ductus arteriosus also must be considered to be a septal defect. It is aortic septal defect, not the classical aortico-pulmonary window or fistula; but it is nevertheless a communication between these vessels. If one classifies it as a septal defect, it becomes apparent that certain logical changes must take place in the lungs if it remains untreated. These changes are arteriolosclerosis of the pulmonary vascular bed. They are the same changes that take place in patients with atrial or ventricular septal defects. Once advanced past a certain critical point, they represent contraindications, at least to our present methods of performing surgery and, perhaps, to any surgery.

In patent ductus arteriosus, it is believed that these changes have gone to the point of irreversibility when a right to left shunt through the ductus has developed. At this stage, the pulmonary vascular resistance will have come to exceed the over-all systemic arterial resistance. Surgical closure of the ductus then merely removes a circulatory safety valve and usually is associated with a shortened period of survival.

It has been estimated by Burwell et al[2] that the existence of a patent ductus arteriosus shortens a person's life by twenty five years. Fortunately, in most cases the lesion is diagnosed sufficiently early for safe corrective surgery. This is a very common way of treating a patent ductus. Four clamps are appplied across the ductus. It is divided between the central two pairs of clamps. These are removed and the vascular cuffs which protrude from the remaining clamps are oversewn with fine silk

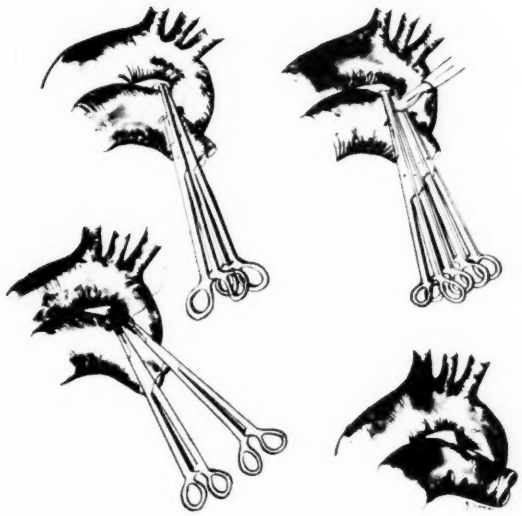

Fig. 2. Application of clamps, division of the ductus, and suturing of the ends.

sutures. It is obvious that recanalization of the ductus (sometimes reported after simple ligation) is precluded with this technic (figure 2).

One of the gratifying things about surgery for a patent ductus arteriosus is that unless advanced pulmonary vascular changes are present, a complete cure is obtained nearly always. The patient goes on the operating table as a cardiac invalid, and he comes off the operating table with essentially a normal circulation.

Sometimes, subacute bacterial infection of the ductus may occur. Fortunately, division of the ductus, plus antibiotic therapy, ordinarily provides a complete cure.

The lesion of the so-called "adult" type of coarctation of the aorta has, at times, been considered as the converse of the patent ductus arteriosus. Skoda[3] presumed that, in these individuals, the tendency toward involution of the ductus (heaviness of the muscular coat) was greater than normal. Not only did the ductus

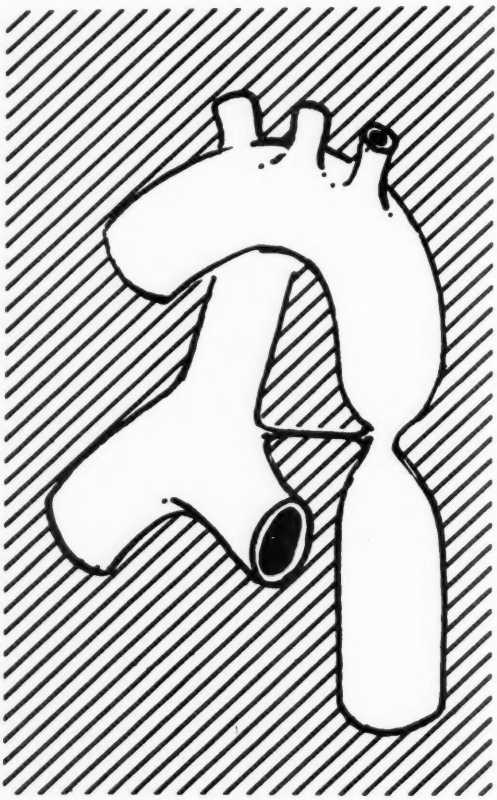

Fig. 3. Diagram of a coarctation or constriction of the aorta.

become involuted completely but that portion of the aorta into which it inserted also became narrowed or obliterated (figure 3).

That is the commonest form of coarctation of the aorta, and one which is associated with a great overdevelopment of the collateral vessels which connect the upper segment of the aorta with the lower segment. These communicating arteries become dilated and tortuous, but are never able completely to replace the function of the main aortic channel.

There are weaknesses in Skoda's reasoning. One of them is that sometimes the ductus does stay open in these cases connecting either with the upper or the lower segment of the aorta. If it connects with the upper segment, it is merely an additive lesion. If it connects with the lower segment, a different anatomical and clinical picture develops.

The legs and lower part of the body are then perfused with pulmonary arterial blood (venous blood). The toenails are frankly cyanosed, whereas the fingers are of normal color. There is no lack of circulation to the lower portion of the body, although the arterial pressure is lower than normal because it is pulmonary arterial pressure. There is no development of a collateral arterial system. Consequently, surgery for this "infantile" type of coarctation carries certain additional hazards.

Perhaps if we had only called in plumbers on this problem of aortic coarctation instead of depending upon doctors to get around to thinking mechanically, we would have solved it long before Crafoord's[4] first successful operation in 1944. He clamped off the aorta above and below, cut out the strictured segment and anastomosed the ends together. Gross[5] duplicated this operation within the same year. This is a clear-cut example of similar advances being made in distant parts of the world, more or less independently (figure 4).

One can clamp the aorta as long as necessary in the "adult" type of disease, because for all practical purposes, it has been clamped all the patient's life anyway. The collateral vessels have always maintained the circulation to the lower part of the body. Hence, these individuals do not get into trouble, even though some of these operations have taken as long as twelve hours. In the "infantile" type of coarctation, in which the ductus supplies the circulation to the lower part of the aorta, there is no collateral circulation. When one interrupts the flow through the aorta by cross clamping, the permissible time limits are short, perhaps less than fifteen minutes. Preferably, general bodily hypothermia should be induced in these patients so that the lower portion of the spinal cord can tolerate the period of ischemia necessary for the performance of the anastomosis.

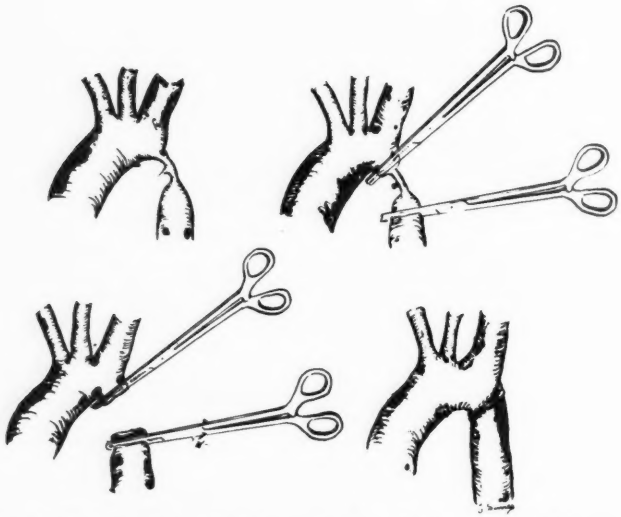

Fig. 4. The application of clamps, excision of the coarctated segment, and repair by reanastomosis of the aortic ends.

Sometimes the area of coarctation is long. Then, after excision of the stricture the ends cannot be approximated. Again, we are indebted to Dr. Robert Gross[6] of Boston for the suggestion that preserved arterial homografts be used to replace the narrowed segment and to restore the aortic continuity. More recently, prosthetic tubes of plastic fabric have become available for this (figure 5).

Of course, there are other problems with the great vessels. One example of this is the double aortic arch, a true vascular ring (figure 6). This condition always becomes manifest in early infancy, because with the growth of the infant the vascular ring constricts the trachea or the esophagus or both. The infant experiences dysphagia or dyspnea, especially after feeding. There are several other arterial anomalies which may amount to a vascular ring and which act in somewhat the same manner.

In this particular patient, the larger of the two aortic arches ran behind the esophagus, while the smaller one ran in front of

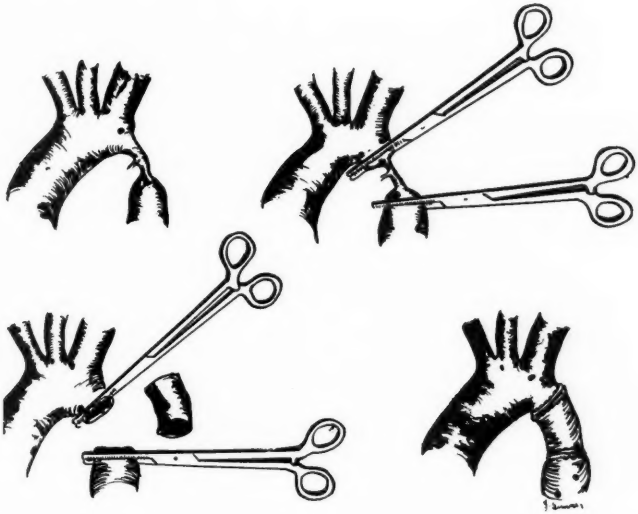

Fig. 5. The technic of insertion of a graft to replace a lengthy coarctation of the aorta.

the trachea. The vessels to the head and upper extremities may arise from but one of these arches, or they may arise in any way. In this case, the problem was simple enough because we divided the ring at the narrow zone, and then bent back the sutured ends of the divided vessels and sewed them actually to the underside of the sternum, so as to open up the ring and convert it into a shallow crescent.

One of the simplest conditions and yet one of the most interesting ones is congenital pulmonary stenosis (figure 7). This may occur as an isolated lesion or it may be part of a more complex group of deformities. When it occurs as an isolated lesion, it is usually valvular. The three cusps of the pulmonary valve are congenitally fused. There may be a slitlike orifice or a tiny central circular aperture. In any case, it is obvious that, even though there usually are remnants of the original commissures, there is only a very tiny passageway for right ventricular blood to enter

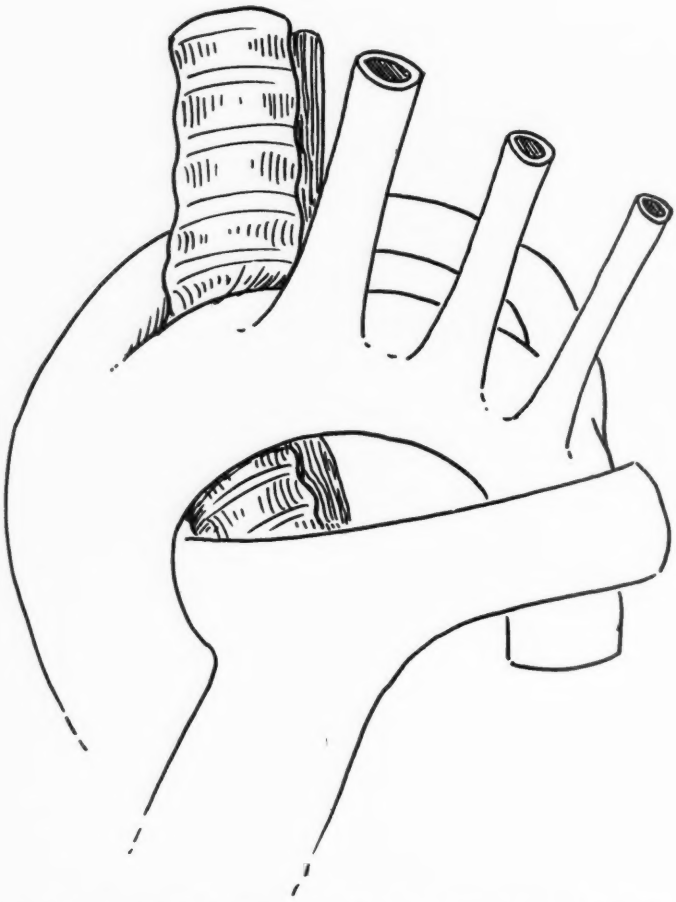

Fig. 6. Diagram of a "vascular ring" (in this case a double aorta) encircling and compressing the trachea.

the pulmonary artery. These cases were hopeless, until the time of the contribution of Sellors[7] and Brock,[8] of London (1948). They devised a closed method of opening up this valve. It was a pretty good operation, although somewhat incomplete. Today, we think in terms of a more perfect operation than we did then.

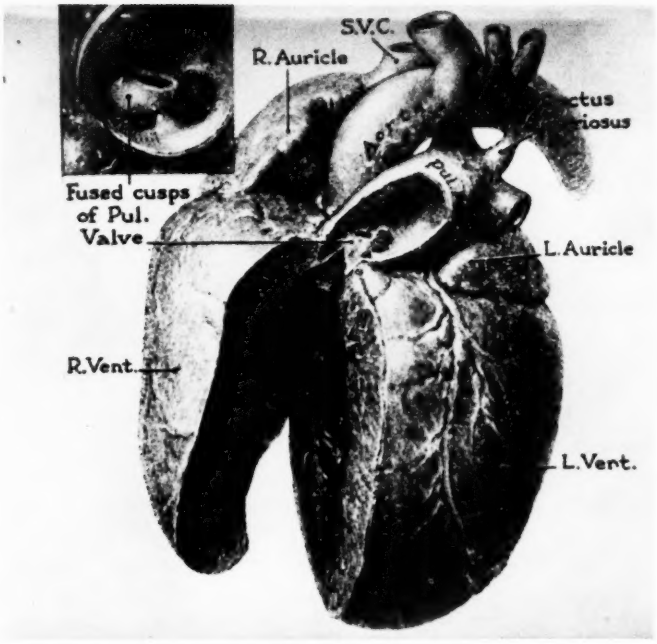

Fig. 7. Sketches of the narrowed pulmonary valve in congenital pulmonary stenosis.

Dr. Henry Nichols,[9] one of my associates and former students, modified Brock's operation and improved it considerably. Whereas Brock would advance a diamond-shaped knifeblade to divide the valve "megaphone" in the hope of producing a bicuspid valve, Henry Nichols modified one of our guillotine knives in such a way that it can be advanced through the valve in its closed position, then opened and engaged upon it (figure 8). The sliding blade cuts all the way to the arterial wall. By using both a right- and left-sided valvulotome, a pretty good bicuspid valve can be created with resultant great reduction in the gradient across the pulmonary valve, frequently to zero.

However, all such "blind" operations have the disadvantage that the surgeon cannot see the incompletely formed commissures.

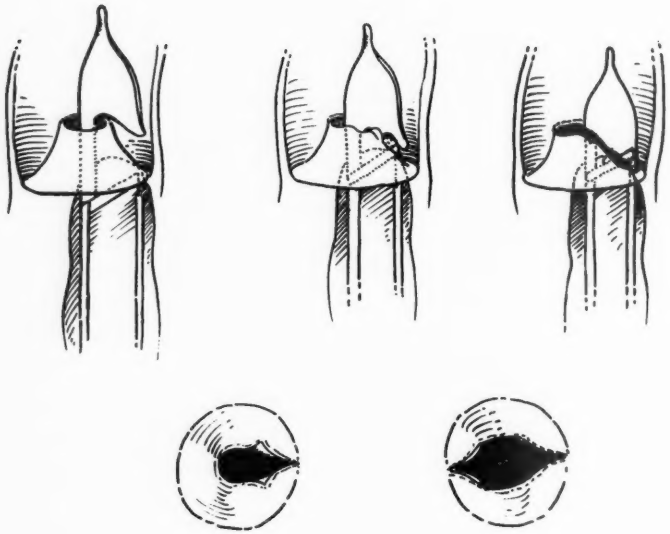

Fig. 8. The relief of the valvular obstruction by the Nichols pulmonary valvulotome.

Consequently, at least one of the rudimentary valve cusps must be divided. Usually, the valve structure is thickened to such an extent that not much regurgitation results. But it is not a perfect procedure; and so, today, this operation usually is carried out by an open technic either with the aid of hypothermia or the "heart-lung" machine.

There is another type of pulmonary obstruction in which there is a ridge of muscle, a noninvoluted or incompletely involuted supraventricular crest which protrudes from the right side of the ventricular septum. It may approach the anterior wall of the right ventricle. This may be associated with a perfectly normal valve, but there is difficulty in getting blood from the right ventricle up to the valve region. Sometimes, this crest is located

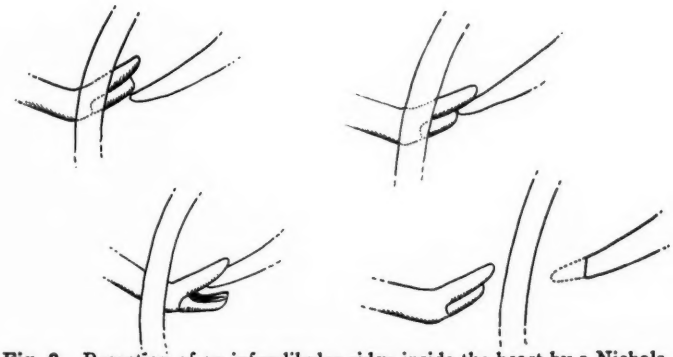

Fig. 9. Resection of an infundibular ridge inside the heart by a Nichols rongeur.

at a low level, an actual additional cardiac chamber existing above it and below the pulmonary valve, the "infundibular" chamber. A small incision is made into the right ventricular wall. A rongeur is inserted to grasp the edge of this ridge of tissue and bite a piece out of it (figure 9). If enough of the ridge is excised, the continuity between the right ventricle and the pulmonary artery is reestablished completely, and the patient becomes essentially normal. If it is but partially excised, a degree of obstruction remains. That is not desirable in patients with a "pure" or isolated pulmonic stenosis; in cases with an associated ventricular septal defect, it is most desirable, because our objective is merely to balance the tendencies toward shunting.

Before discussing the more complicated lesions, I would like to describe the simpler intracardiac septal defects, or, rather, atrial septal defects. One patient with a moderate-sized lesion of this type was a woman, 43 years of age, who came to me in December 1951, seeking help for an atrial septal defect, diagnosed by cardiac catheterization. This lesion had caused her to be an invalid for many years, and had led to heart failure which was more or less persistent during the previous two or three years (figure 10).

At that time, we had no real operation for the condition. The nearest thing to an effective operation was Dr. Henry Swan's[10] attempt to invaginate the two atrial appendages against the defect,

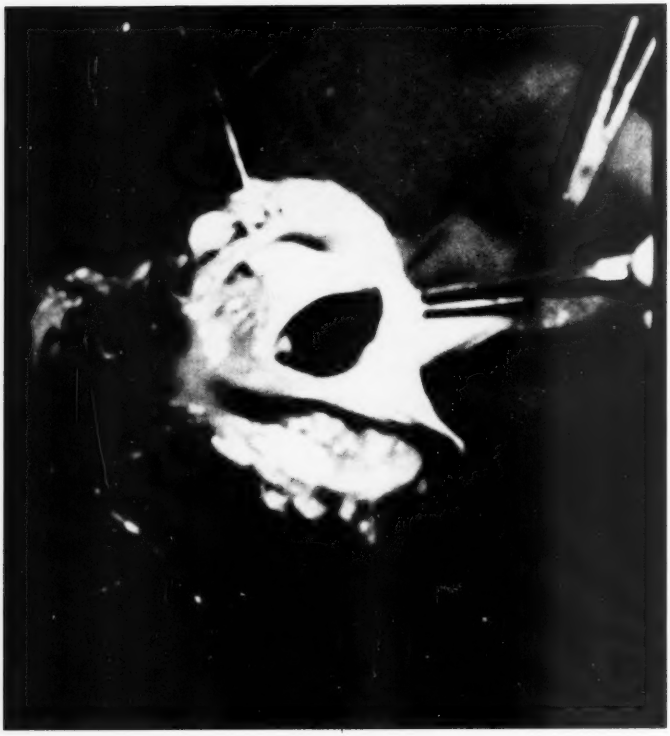

Fig. 10. Photograph of the inside of a heart with an interatrial septal defect.

but this procedure had not been proven effective at that time. Since then, it has been disproven.

Having some doubts about the outcome of such an attempt, I suggested that she go home and spend the holidays with her family and return in January of 1952. She came back and my presentiments were well founded because she died during the induction of anesthesia before we had a chance to do anything definitive for her heart.

After she died, her husband and her son urged me to perform

an autopsy in the hope that something might be found which would help others with a similar condition. Never was an unselfish and charitable purpose more completely satisfied! I was unable to sleep for thinking of this poor woman and this lesion which, obviously, was a very simple one. There was a lack of about five grams of cardiovascular tissue, and that was all. Otherwise, she had a normal cardiovascular system. A deficiency of one sixth of an ounce of cardiovascular tissue, and she had to die at an age which some of us think is quite young!

On the third wakeful night, at about three o'clock in the morning, it suddenly occurred to me that this problem could be presented in terms of elementary algebra. This negative quantity of cardiovascular tissue may be considered as minus X; and, in order to solve a hypothetical equation, all that would be necessary would be an equal amount of positive cardiovascular tissue, excess tissue, which could be applied directly over the defect. Then, the equation $-X + X = 0$ would be satisfied. With that beginning and within the day a feasible technical solution[11] was devised. The greatly dilated right atrial wall represents an excess of cardiovascular tissue which can readily be compressed down against the septum, and can be sutured to the margin of the defect, thus closing it physiologically, and separating the two venous streams. A finger may be inserted through the right auricular appendage to feel the defect, and sutures may be passed under the guidance of the intracardiac finger to approximate the lateral atrial wall to the margin of the defect. Eventually, the globular right atrial chamber is converted into a doughnut-shaped chamber which still has a larger than normal capacity. Therefore, there is no significant interference with the passage of blood to the tricuspid valve. The defect is patched permanently with normal cardiovascular tissue (figures 11A-D).

Within two weeks, a woman from Cleveland came to us with a similar lesion. She had been in heart failure for most of the previous two years. At the completion of surgery, the remnant of the once greatly dilated right atrium showed as a dimpling in or invagination of the right atrial wall. It subsequently was proved by catheterization that we had achieved complete closure of the defect. Apparently, this is the first historical example of closure of an interatrial defect by any technic (figure 12).

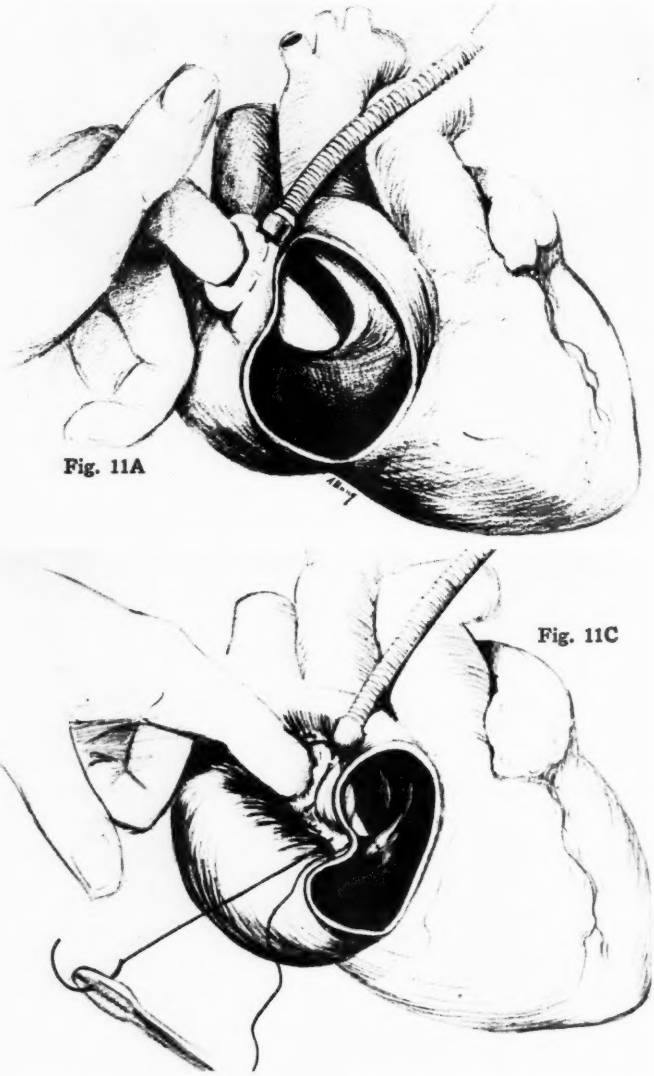

Fig. 11A

Fig. 11C

Fig. 11. (A, B, C, D). The exploration and technic of closure of the atrial septum through the purse-stringed atrial appendage.

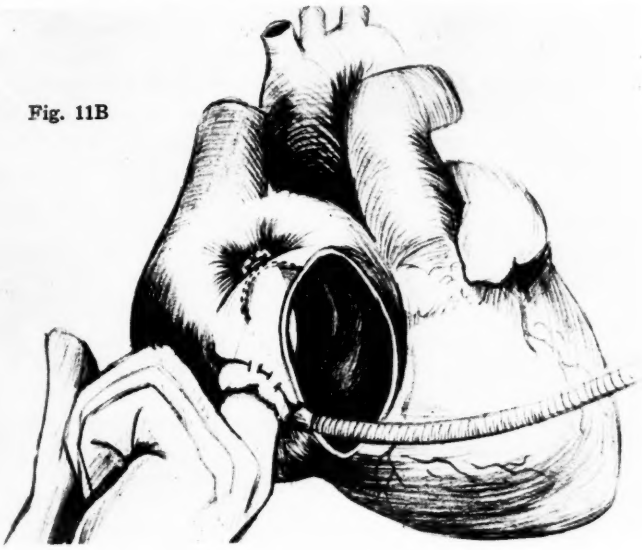

Fig. 11B

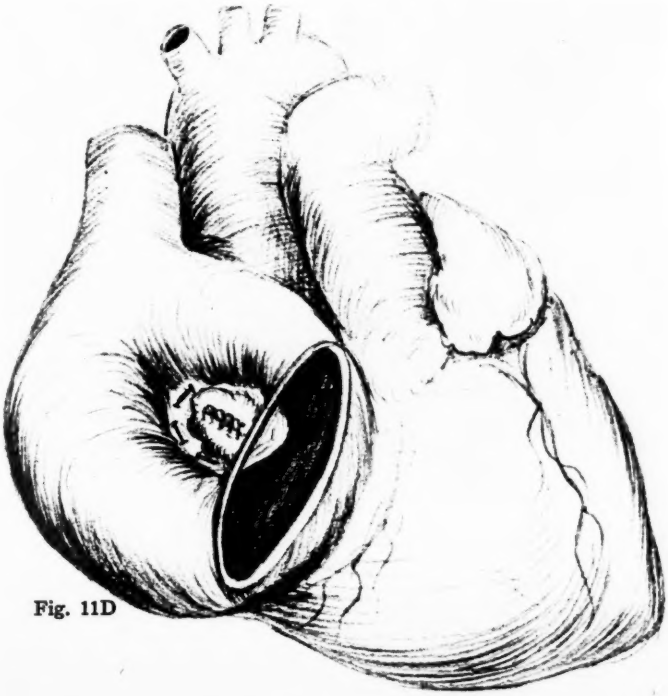

Fig. 11D

We have continued to use this method which has been called "atrioseptopexy",[11] and have found it to be most useful, particularly in young people, such as children, with secundum type defects. Occasionally, we encounter anomalous drainage of the right pulmonary veins into the right atrium, but this is no particular problem. One of my associates, Dr. Wilbur Neptune,[12] solved the problem of their correction. After recognizing the situation by intracardiac palpation, chaotic thoughts of removal of the normal right lung came into mind, because if the defect were closed and the right lung permitted to drain into the right atrium, the physiological abnormality would remain. The thought of removing a normal lung in a young woman was repugnant, yet, what were we to do?

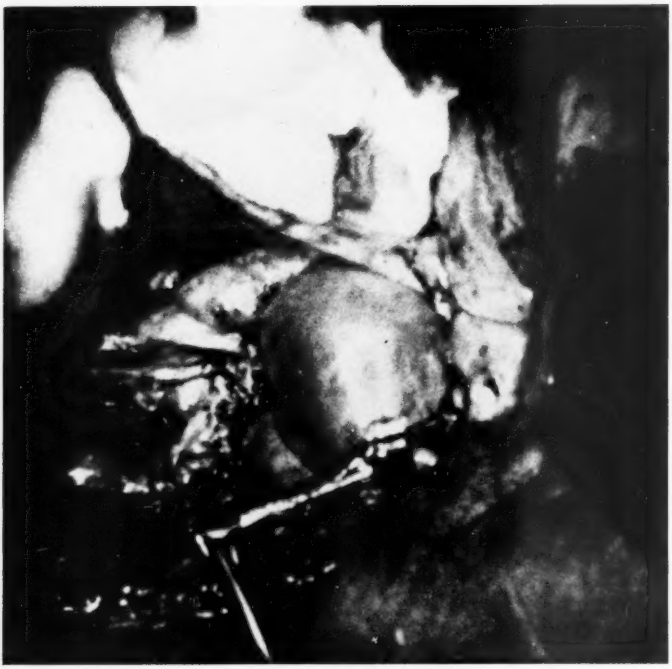

Fig. 12. Photograph of the "dimple" in the external wall of the right atrium following atrial septal defect closure.

Dr. Heiser, the old "horse and buggy" doctor, has said that the main function of the mask in the operating room is to keep people from knowing when the surgeon is chagrined, embarrassed, or frustrated. All of those represented my state of mind. Each and every one of my assistants was permitted to feel the lesion, hoping that one of them could suggest a useful idea. Usually at such a juncture, no one has any helpful thoughts. But this time the last one, Dr. Neptune, after he felt it said, "Oh, that's good! All you have to do is sew the lateral wall of the atrium to the anterior margin of the defect, and you'll retranspose the veins at the same time that you separate the venous streams."

I did not quite see it but did have the mask to protect me, so queried, "Well, Doctor, how would you propose to do it?" So he told me. And so I did it, and the veins of the right lung then emptied into the left atrium, yet the venae cavae and their circulation were entirely separate from the left atrium (figures 13A, B).

We have done approximately a hundred and fifty of those operations now, and the mortality in properly selected cases is less than five per cent. The cure rate, as proven by subsequent catheterization, is in excess of 95 per cent. Occasionally, a stitch cuts out or we do not put enough in, and then there is some residual shunt, but that is not very common.

The operation can be done quickly and easily and with a minimum of trouble. However, in a complicated lesion such as an ostium primum defect, a total absence of the atrial septum, multiple defects, or certain other peculiar situations, an open technic may be necessary.

There are two ways of operating on these cases under direct vision. The first is with hypothermia. The bodily temperature is lowered to permit occlusion of the venae cavae for a longer period than is permissible at normal temperatures. The atrium then is opened revealing the defect. The edges may be sutured together directly, or a patch of plastic material such as Ivalon sponge may be sewn to the margins of the defect. These x-rays of the first surviving patient show the progressive reduction in the size of her heart and the diminution in the size of the pulmonary vessels after correction of the lesion (figure 14).

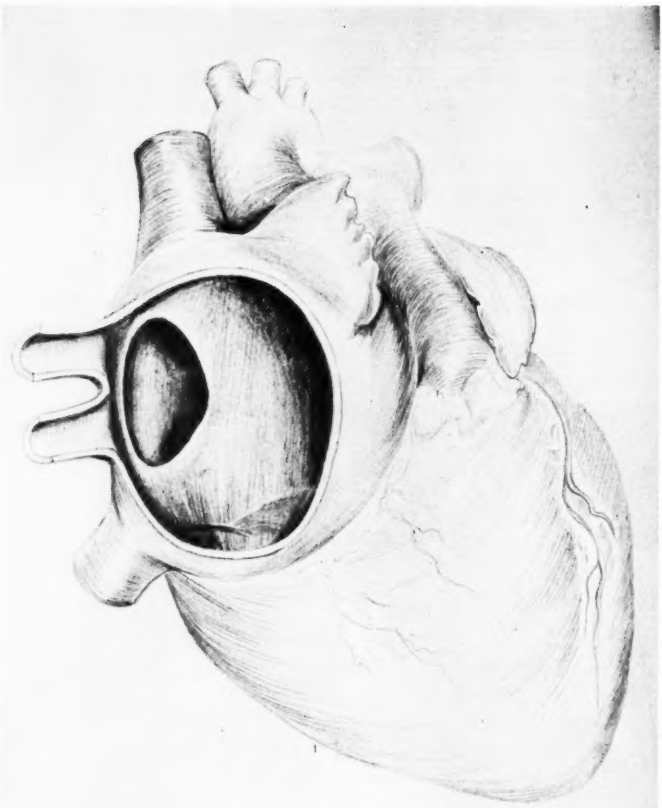

Fig. 13 A

It should be mentioned that, of course, the flow through an atrial septal defect tends to be from left to right. If there is no complication, and as long as the pulmonary vascular bed is of normal resistance or at least not too greatly damaged, that direction of flow will pertain. Gradually, with the continuous engorgement of the pulmonary vascular bed, over the years, changes tend to take place in the pulmonary arterioles just as in patent ductus arteriosus. Consequently, the media thickens, the intima undergoes

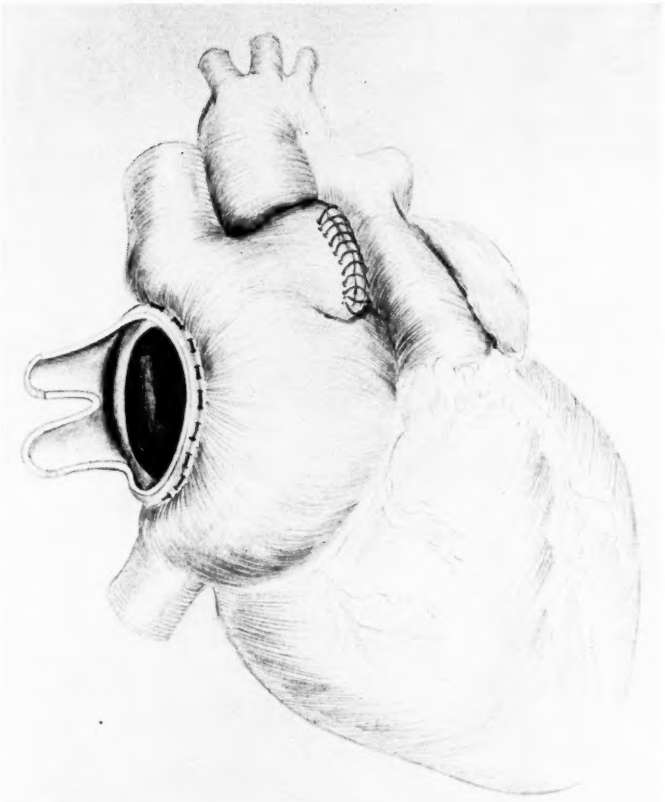

Fig. 13 B

Fig. 13. (A, B). Anomalous pulmonary veins (on the left side of the diagram) entering in juxtaposition to an atrial defect, and the correction of both defects.

hyperplasia, and ultimately fibrosis or thrombosis of these vessels may take place. At first, there is a considerable element of spasticity in these arterioles which causes the pulmonary vascular resistance to rise. Gradually, the pulmonary arterial pressure rises which makes it harder for the right ventricle to empty itself. The

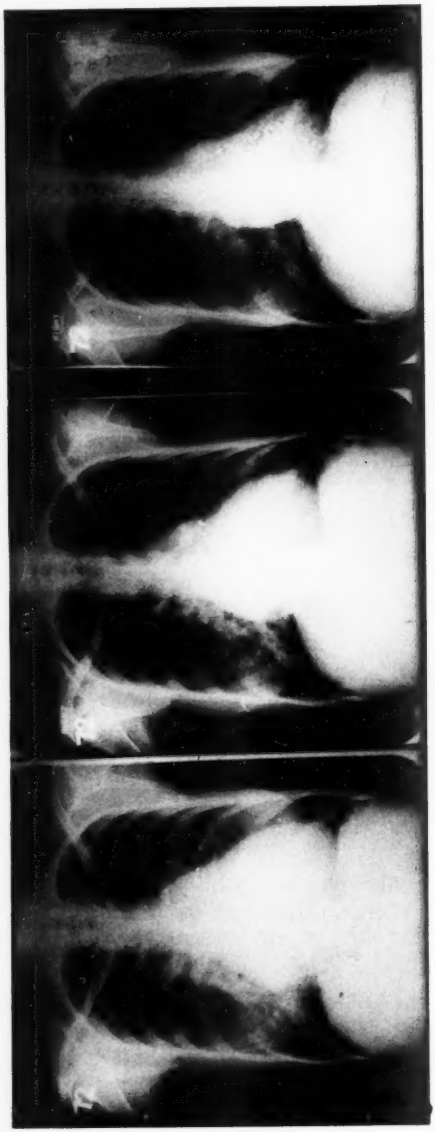

Fig. 14. X-rays of the first surviving patient showing progressive decrease in the heart size following closure of an atrial septal defect.

time comes when the right ventricle either fails or is on the verge of failure. At that time, it does not empty itself completely and, because it retains considerable blood within its chamber, it becomes difficult for the right atrium to empty its contents into the right ventricle against this backlog of contained blood. Therefore, some of the right atrial content spills over into the left atrium causing a degree of right to left shunting.

There comes a time in this disease, therefore, as in patent ductus arteriosus, when true reversal of the shunt takes place and unoxygenated blood gets into the left side of the heart and, hence, into the aorta. This is manifested by undersaturation of the arterial blood and, perhaps, by actual cyanosis. At this point it is generally believed that the pulmonary vascular changes have become irreversible and that surgery is contraindicated. I do not know whether this concept is entirely sound. Recently, we have been operating on these patients, using a valvular type of plastic patch called a "toilet-seat" valve. This permits a gradual closure of the defect over a period of months, thus allowing gradual adjustment of the circulation.

One can operate under direct vision if the bodily temperature is reduced sufficiently. How much is this? Well, if the patient is cooled to 85°F, there are seven or eight minutes at least during which one can operate with the venae cavae obstructed. If one perfuses the arterial system with heparinized blood during that time, it is possible to double the permissible period.

At 85°F the ventricles are not very irritable, and the heart is relatively easily resuscitated. There are many ways of inducing hypothermia. Immersion in ice water or packing in ice is effective. In either instance, it is necessary first to induce general anesthesia. Considerable time is required — perhaps five hours in an adult.

The matter of spending a half hour to establish a hypothermic state is acceptable, but spending five hours of the anesthetist's time is excessive. Furthermore, it is not good for the patient to be under anesthesia all that time.

Moreover, one must be ready to open the chest at any time because the heart may develop ventricular fibrillation suddenly. Constant monitoring with the electrocardiograph is essential.

Another disadvantage is the uncertainty regarding the desirability of hypothermia until the cardiac pathology has been explored. Secundum defects of the interatrial septum are readily repaired by the technic of atrioseptopexy. But should the defect be of the ostium primum type, an open technic using hypothermia may become mandatory. Induction of hypothermia after a large incision has been made in the chest presents many problems.

Dr. Brian Blades[13] of Washington has made a very practical contribution. He suggested that one may set up a continuous system, whereby iced saline solution is poured into one side of the chest and aspirated from the other. It is then passed through a cooling coil and reused. By this method, an adult patient may be cooled to 85°F within an hour (figure 15).

There are a great many different types of useful heart-lung machines today, and if for some reason the surgeon does not wish to use hypothermia, he may use the heart-lung machine. Some think that is the preferable method. Certainly, it gives one more time which might conceivably be needed for the closure of an atrial septal defect.

With ventricular septal defects, we do need more time. Furthermore, hypothermia is undesirable when the ventricle is to be opened because, when wounded, it tends to go into ventricular fibrillation if the temperature has been lowered enough to permit a reasonable period of time for intracardiac surgery.

It should be said, before going on to ventricular septal defects, that there are other technics for performing closed surgery for interatrial septal defects. Dr. Robert Goss[14] of Boston has suggested a method whereby a "well" of latex can be sewn to an incision made in the right atrium. Then, using local heparinization, the operator's fingers may be dipped through this well to feel the defect. One may sew a patch of tissue or a patch of plastic sponge to the margin of the defect.

During recent months and years, he has been using our somewhat simpler technic in most cases, but Dr. Kirklin[15] of the Mayo Clinic still uses the original Gross well technic for suturing atrial septal defects.

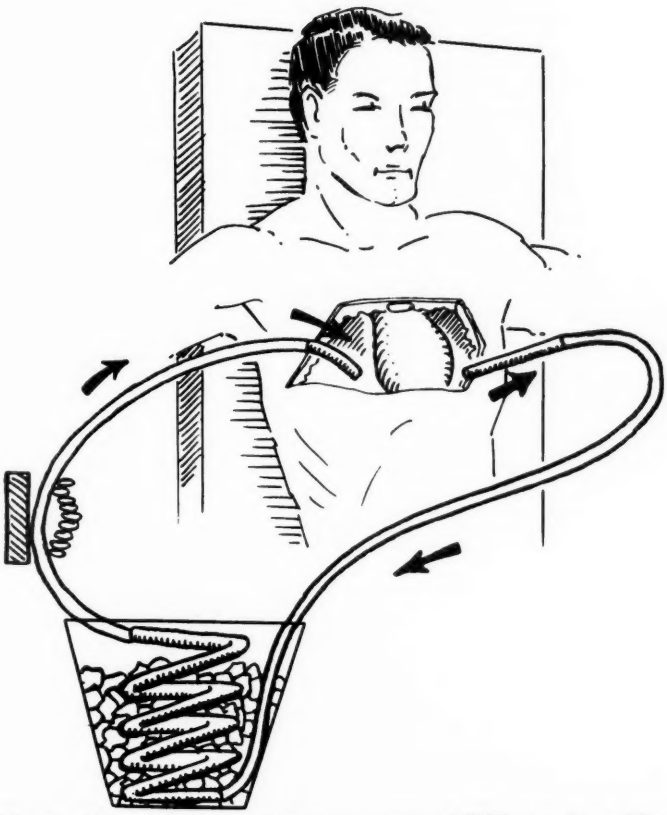

Fig. 15. Hypothermia to a body temperature of 80°F. may be rapidly induced by irrigation of the open chest with cold saline (Blades).

Another method is that of Søndergaard et al.[16] He has worked out a way of placing a purse-string suture all around the margin of the atrial septum. When the suture is pulled down tight, it puckers the septum and apparently, in many cases, obliterates the defect completely.

However, in ventricular septal defect, there is no satisfactory

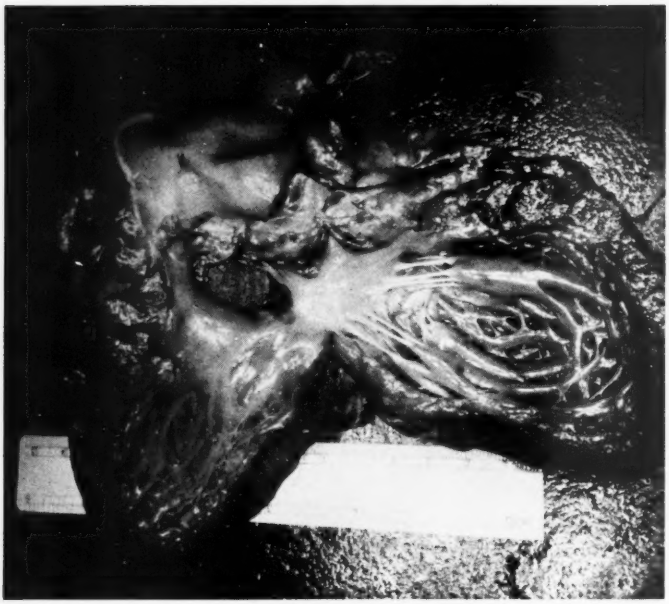

Fig. 16. Photograph of a high septal defect lying between and below two of the aortic valve leaflets which had been closed with an Ivalon patch.

closed surgical technic and it does not appear that there ever will be. For one thing, the defects vary greatly in size and location. Small ones can be closed with a couple of stitches; however, the defect may be larger. A large ventricular septal defect, showing an actual loss of substance, usually is found high, involving the membranous portion of the septum (figure 16). For this particular defect, the probabilities are that one would have to use a patch of prosthetic material or a tissue graft in order to close the opening permanently.

Now, thanks mainly to the pioneering of Dr. Clarence Lillehei[17] of Minneapolis, we now have a method of closing ventricular septal defects, using total cardiopulmonary by-pass. His original method of using a human donor is no longer considered acceptable, but we can perfuse the patient with many different types

of artificial equipment. The venae cavae are individually cannulated. A "Y" tube connects the streams of venous blood together, and they are pumped into an oxygenator which can be of several different types. The blood is drained from the oxygenator and is propelled by a second pump into one of the great arteries, usually the left subclavian, although the femoral or right subclavian may be used. Then, of course, the only connection of the heart with the circulation is through the coronary system and through the bronchial vascular system. One may usually ignore this small amount of blood and open the heart and operate in nearly a dry field. "Nearly" is an appropriate term (figure 17).

In the tetralogy of Fallot, one has two problems. These are, of course, an intraventricular septal defect and also pulmonary stenosis. The pulmonary stenosis has received most emphasis in

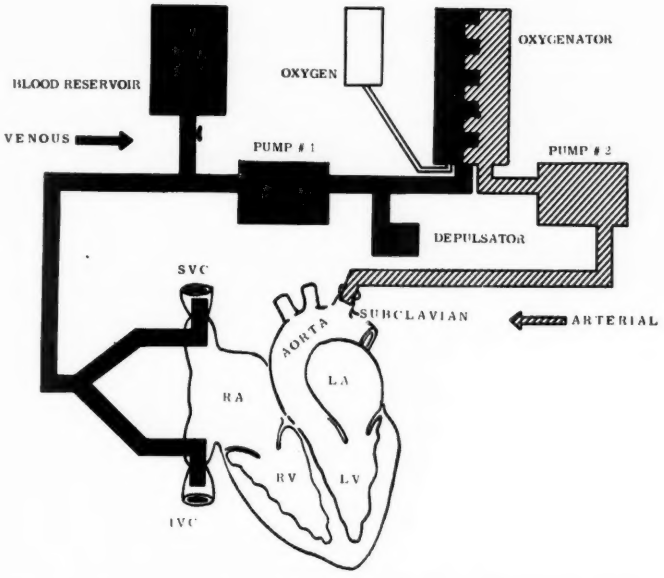

Fig. 17. Diagram of the circulation of the blood through the pump oxygenator. The venous blood is depicted black, the oxygenated "arterialized" blood by the shaded portion.

the past and quite rightly so, because it prevents the venous return from entering the lungs to be oxygenated.

The contribution of Dr. Alfred Blalock[18] to this work is well known — the creation of an artificial patent ductus by anastomosing one of the subclavian arteries to a pulmonary artery in end-side fashion (figure 18). The communication permits a left to right shunt to occur. This acts largely to balance the right to left shunt which occurs through the septal defect. The individual's color becomes less blue, and more importantly, he becomes able to perform exercise whereas previously he could not. Of course, this operation does nothing for the intracardiac lesions. However, it was a good operation, although often but a palliative procedure. Certainly, its application has permitted many individuals to survive to the era of definitive correction of these conditions who would not otherwise have survived. It still has real therapeutic value, especially in certain complicated cases.

Sir Russell Brock[19] of London suggested in 1950 that in the tetralogy of Fallot the obstruction to the pulmonary artery or the infundibular obstruction be relieved directly, and for many years

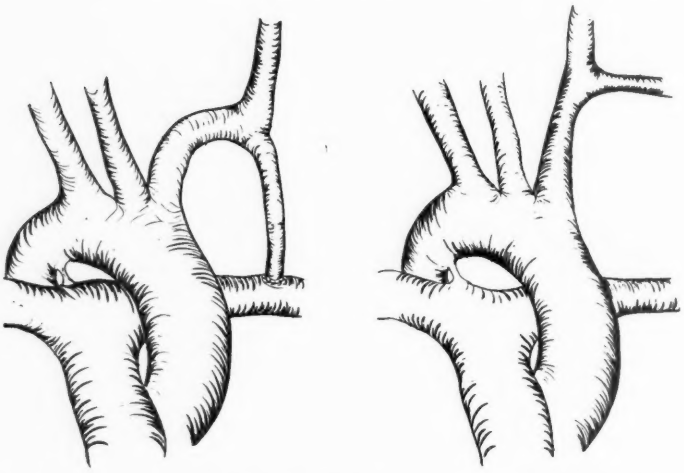

Fig. 18. The similarity of the Blalock-Taussig operation (on the left) to the condition found naturally as a patent ductus (on the right).

we have practiced that operation, thinking it generally better than the shunt operation of Blalock (see figure 9). However, it is only half an operation because with relief of all the pulmonary obstruction, the patient develops a left to right shunt through the defect, just as in isolated ventricular septal defect. Therefore, it is necessary to limit the amount of relief of the pulmonary obstruction in each case to one quarter or one half. Nevertheless, the patients routinely are improved.

If this operation were ideally done so that the two ventricular pressures were balanced, there would be no shunt through the defect, and the patient would be restored to relative normality, although his right ventricle would remain in competition with the left.

That operation should not be considered obsolete today, because if one operates definitively for the tetralogy of Fallot, the mortality is very high. If the patient is an infant under two years of age, the risk is particularly great. The reasons why these people are likely to die are several. One of them is that the blood circulating may have become more or less incoagulable, especially in older people. In all cases, the extensive collateral blood supply leads to excessive operative bleeding. In infants, there is not enough respiratory reserve to tolerate transection of the sternum and surgical opening of both pleural cavities.

In any case, the pulmonary arteries are likely to be hypoplastic and incapable, therefore, of accepting a normal flow of blood. Congestion and hepatization of the lungs are common after definitive surgery. In a deeply cyanosed individual and in an infant, it is preferable to permit the lungs to become adjusted to a greater flow by performance of a Blalock shunt as a controlled Brock procedure and then, a couple of years later, do a definitive operation. Also, in patients with ventricular septal defect, in whom the pulmonary arterial pressure has risen to more than two thirds of that in the aorta, the operative mortality has proven to be quite high with the corrective procedure — 40 or 50 per cent. In individuals with a lower right ventricular pressure, the surgical mortality is relatively low, less than 15 per cent.

If a patient with a ventricular septal defect is a small infant,

or if the pressure in the pulmonary artery is over two thirds of that in the aorta, we consider that the vascular changes which have taken place as a result of the prolonged left to right shunt are such as to preclude safe operation by the open fully corrective method. In these cases, we do what might be considered the reverse of the Brock procedure.[20]

We clamp the pulmonary artery partially, and measure the pressure distal to the clamp. The amount of narrowing is adjusted until the pressure is reduced to 25 to 30 mm. Hg (a normal pressure). Then, the vessel is sutured to maintain that much constriction of the artery. If there remains any significant degree of reversibility in the pulmonary vascular lesions, regression of those changes may take place. Later, the patient may improve to the point where an open operation may be done and all the lesions be corrected with a very small operative risk. We are going to have to individualize all these cases in the future; otherwise, we will never be able to get the curative procedures to the level of safety where they should be.

In operating on a congenitally stenosed aortic valve today, it is believed that an open technic is preferable to a closed one because it permits visualization of the rudimentary valve cusps (figure 19). The rudimentary commissures must be separated with critical accuracy. By visual guidance, the surgeon can avoid cutting across a valve cusp. This would be harmful to the patient if it were a pulmonary valve and would be lethal if it were the aortic valve.

Hypothermia may be used for either of these operations. They are short operations and it is not necessary to stimulate or incise the ventricle, so there is really no great objection to this method. We have generally preferred to use the heart-lung machine in both types of cases, but either method affords direct vision of the valve.

Here is, essentially, what is done. The valve is divided accurately along its commissures (figure 20). These leaflets are flexible. They do not contain calcific salts and usually function quite well after separation. Perhaps I may be forgiven for contrasting congenital aortic stenosis with rheumatic aortic stenosis which is a very different thing. Here, the leaflets progressively grow to-

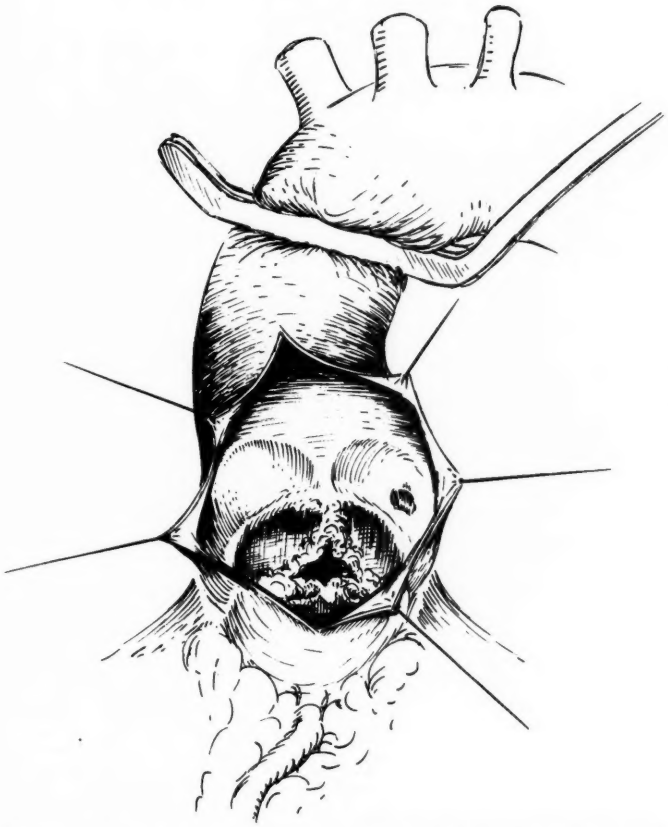

Fig. 19. Sketch of the transaortic exposure of the aortic valve using a bypass.

gether, starting at the aortic wall. Eventually, the aortic orifice is reduced to a tiny central triangle. However, it does not always become a triangle (figure 21). Sometimes, one commissure will obliterate completely, leaving a bicuspid valve (figure 22). Occasionally, two will obliterate completely, leaving a radiating slit. There is a great tendency toward the development of calcification in this valve (about 90 per cent of the cases).

Our original operation consisted of passing a triradiate dilator

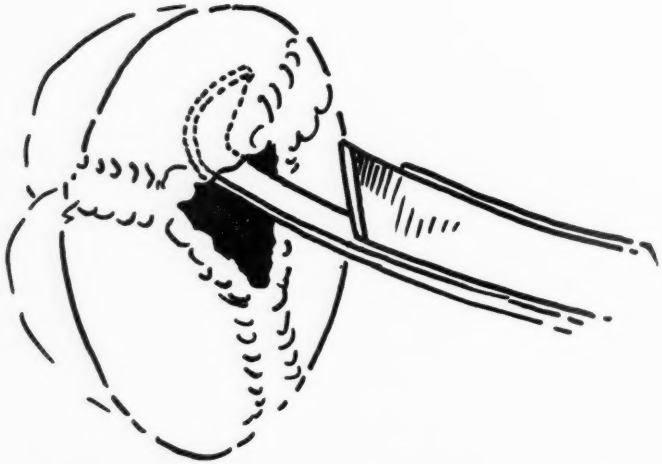

Fig. 20. Accurate division of the aortic commissures by a guillotine-type knife.

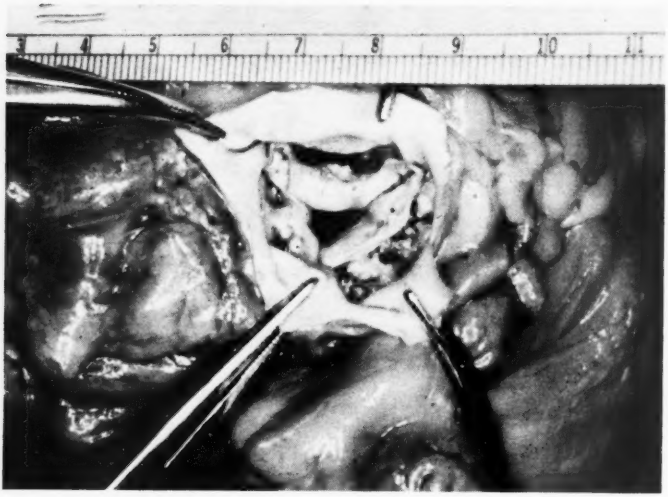

Fig. 21. Photograph of the triangular opening in a stenotic aortic valve.

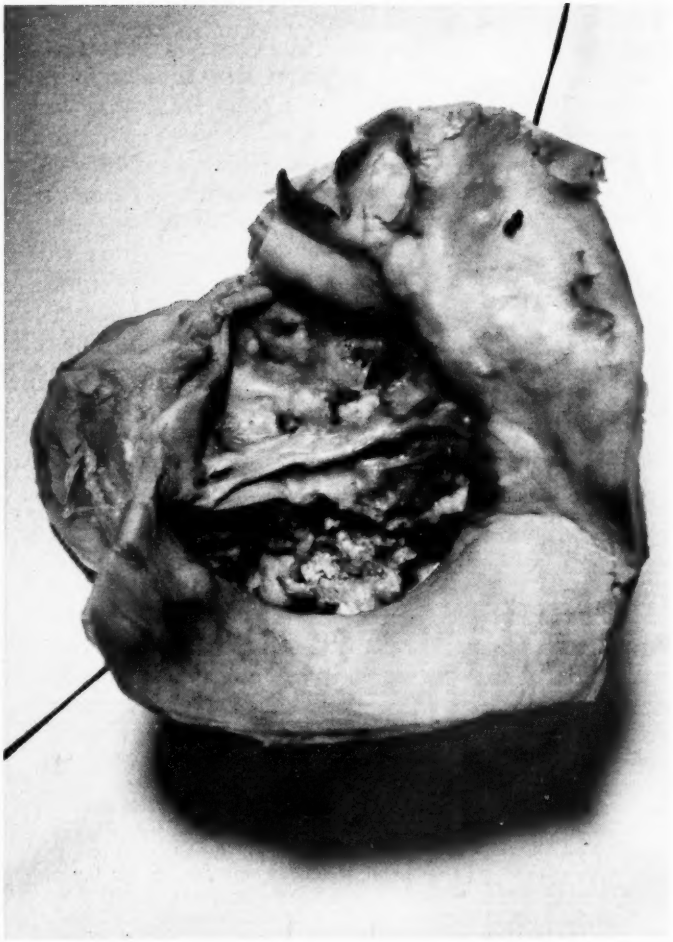

Fig. 22. Photograph of a bicuspid (congenital) aortic valve.

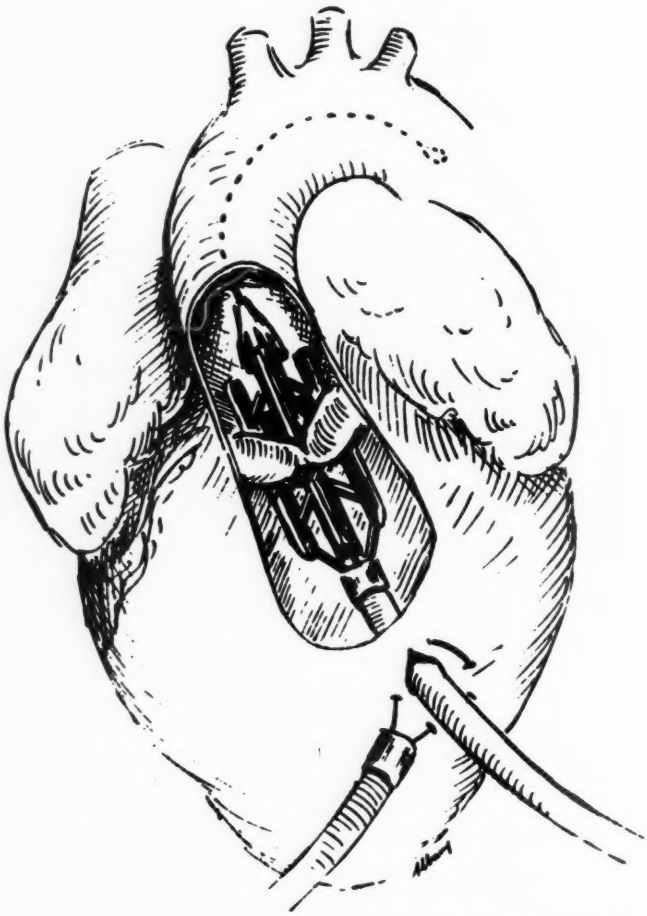

Fig. 23. The triradiate dilator introduced through the left ventricular wall, and then opened. (This blind technic is inferior to the open approach.)

through the ventricular wall and diminutive aortic orifice, compressing the instrumental handles, and applying pressure against the respective commissures. Sometimes, this method worked well and these people were greatly improved. Sometimes, unfortunately,

the commissures would not split and, since something had to give, a cusp would be torn, producing regurgitation (figure 23).

We, therefore, abandoned that procedure, and now with the availability of the open technic have no doubt about the proper approach. We use a heart-lung perfusion system, stop the heart, with potassium or acetylcholine, and then, after clamping the upper aorta, open the aortic root and look at the valve. The appropriate operative technic in any given case is not just a matter of cutting the commissures. We have learned a good deal about this disease since it has become possible to see the valve during life. Sometimes, there are excrescences of calcium over the valve orifice that can be picked off with a pair of forceps, or curetted or bitten off with rongeurs

Occasionally, one encounters an essentially bicuspid valve. The obliterated commissure and the two fused leaflets may present a calcified ridge or diaphragm. Sometimes, an attempt to cut a commissure which is almost totally replaced with calcium will lead to flaking off or fragmenting into sand. The sand will fall away leaving two cusps each with but one side. This causes a very severe regurgitation and perhaps immediate death.

Sometimes, one leaflet will be found to be much smaller than the other two, due possibly to chronic distortion and shrinkage. It is not only small, but is no longer deep and cuplike. It is a flat triangular piece of leather-like material that protrudes from the side of the aorta and is attached to one or both of the other leaflets. If it is detached from such adjacent supports, it will be unable to restrain backward flux of the aortic stream and severe incompetence will ensue. Therefore, the only cusp that may be mobilized with safety is one that is deep and will hold blood.

Occasionally, one finds this situation. All the commissures have been opened, but the valve remains stiff and the rigid leaflets do not move. They are still in close contact. One cusp is rigid and flat, another is also rigid and flat. They should be left as they are because anything done with them will make them incompetent.

However, the third cusp is deep and, while it is rigid due to calcific infiltration, it will hold water. Since the calcification

ordinarily involves only the concave surface or face of the cusp, it may be removed by a technic of "thinning" or sculpturing. The convex surface after such mobilization will be found not only to be flexible, but also appreciably *longer*. It might be considered a unicuspid valve, but a least it will function pretty well. The valve will not be normal, but the mobilization has been a step in the right direction.

Manometric tracings show the typical gradient between the left ventricular pressure and the aortic pressure, which is pathognomonic of this disease. This may be abolished when the patient has been operated upon successfully. If but one cusp is mobilizable, the two others remain as a rigid shelf across the valve and, of course, there will be residual impedance to blood flow. But, the gradient will have been reduced considerably. Furthermore, the patient will have an aperture which is, perhaps, ten times as large as it was prior to surgery; and such an individual will be very much better clinically.

Sometimes, there is aortic stenosis; but there also is severe regurgitation, perhaps more than was anticipated. After the commissures have been mobilized, the leak is just as great as before. Obviously, then, that is the major problem to the patient. What can be done about it?

It is well known that aortic regurgitation has been a very severe problem to the vascular surgeon in the past. Many workers have tried different methods and have not succeeded. At last, the correct answer seems to be near. Simple "sculpturing" of the leaflets as described may provide sufficient lengthening of the cusps to overcome the incompetence. One leaflet may be excised completely. If the other two leaflets are relatively flexible and cuplike so that they can hold blood, then, under direct vision, mattress sutures may be placed across from commissure to commissure, thus plicating one third of the aortic valve annulus. Thus, we convert this into a bicuspid valve (figure 24). If the other two leaflets retain significant mobility, it will be a functional bicuspid valve.

This operation has not been done on a great many patients, but is used in this particular situation and it is amazing how well they

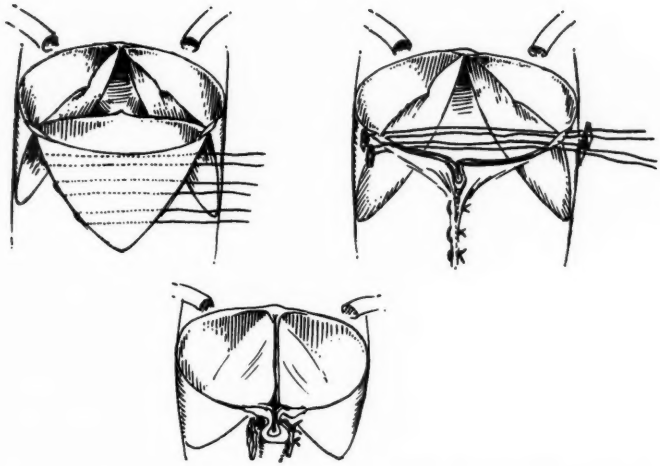

Fig. 24. The correction of aortic insufficiency by conversion into a bicuspid valve.

do. Four ninths of the original cross sectional aperture remains and they have a competent valve.

Of course, that led to consideration of congenital aortic regurgitation, or at least regurgitation which apparently is due to a "weak annulus"; and so attempts were made to correct some of them. In this case, the root of the aorta is dilated and normal cusps can not approximate in the center. That is why these people have aortic insufficiency. By putting mattress sutures across in the same fashion as mentioned previously, converting it to a bicuspid valve, two thirds of the valve leaflet substance is preserved while the valve aperture is reduced by five ninths. That is a mathematical advantage which is quite effective in most cases of aortic insufficiency.

In one of these patients, the blood pressure in the brachial artery showed an average of 160/35 mm. Hg before the operation. After the procedure, there was a remarkable change with a pres-

sure of 148/92 mm. Hg, and a pulse tracing which would be considered quite within normal limits.

Surgery for the correction of mitral stenosis known by the terms "commissurotomy", "valvuloplasty", "valvotomy", or "valvulotomy", as done throughout the world today, is only a step in the right direction. It is not the right or full answer to the disease.

The problem in mitral stenosis is that the circum-orificial centimeter of both leaflets is converted into leathery scar tissue. At the same time the leaflets grow together at either extremity and so the aperture which is normally a semicircle becomes reduced to a small almost straight segment of the original great arc (figure 25).

The result is that we have a linear slit which has been interpreted as the distal end of a flutter valve. Actually, it is just a small segmental arc of that original semicircle. The mural leaflet, the concave leaflet of the mitral valve, measures only a centimeter from free margin to base and so, in most cases, it is totally destroyed by the rheumatic process. It is converted into leather, or stone, or tissue resembling these. When first seen, these people with mitral stenosis have a little slit, the leaflets having grown together and the mural leaflet having become so thickened as to be irreversibly stiffened. It cannot be mobilized again. The septal leaflet, on the other hand, if mobilized, is only

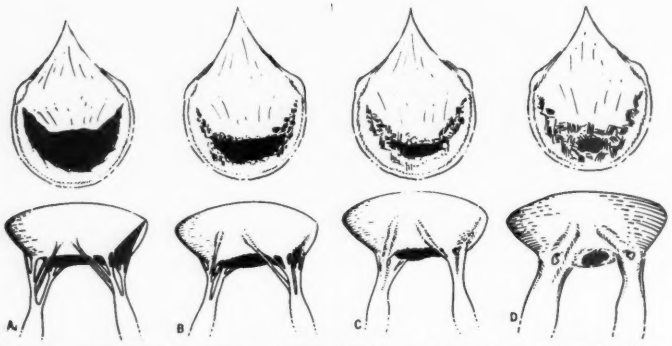

Fig. 25. Progressive stages in the fusion of the mitral valve leaflets above and the subvalvular supports below.

stiff in its distal third. It is flexible in the middle and at its base. Being convex in outline, one can readily mobilize it in such a way that it will function.

Unfortunately, the left-sided approach which we have used over the years does not lend itself well to complete correction of the stenotic mitral valve. The surgeon must work with his index finger bent backward. Any instrument used must also be equally bent or curved. If the mitral valve is approached from the right side, one can work with a straight finger or a straight instrument. All of the inherent technical problems are, thereby, greatly reduced (figure 26).

This is our approach. The interatrial groove is dissected. The term "left atrium" is a misnomer. It is the posterior atrium. A special pair of scissors is slid down the operator's finger into the dissection to make an incision into the left atrium. The finger is wriggled into the opening tamponading it (figure 27).

It is advanced to the valve and by digital pressure, or by the use of an instrument, the entire normal semicircular orifice of the valve is reestablished in at least 75 per cent of all cases.

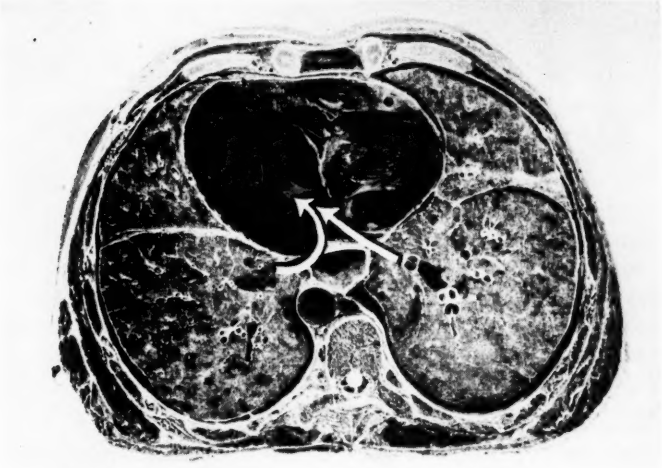

Fig. 26. The two approaches to the mitral valve: from the left side (curved arrow); from the right side, the preferred technic (straight arrow).

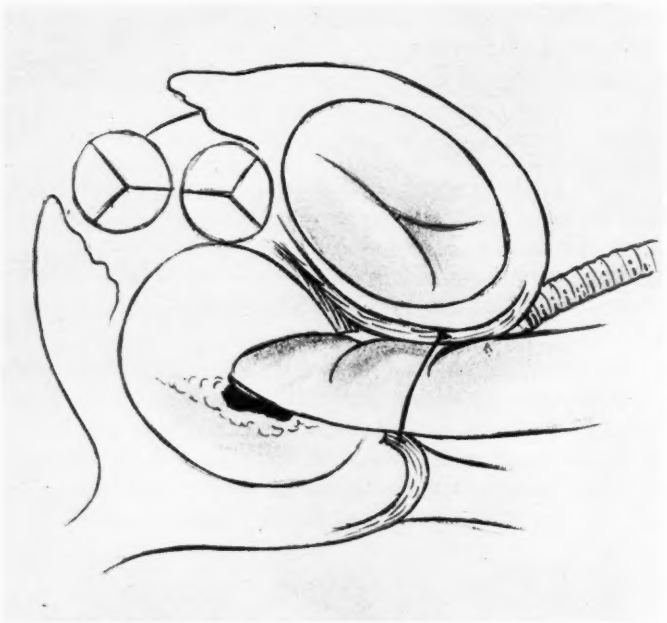

Fig. 27. The finger is introduced through a purse-string suture directly into the mitral valve when the right thoracic approach is employed.

Instead of extending the valve "slit" in a linear fashion to the annulus, as with the old operation, thus necessarily dividing the substance of the mural leaflet at two points, we now reestablish a natural line of valve cleavage[21,22,23] carrying it beyond the first site of leaflet cross fusion, or a little farther than normal. Since the septal leaflet is quite flexible in its midzone, it then will bend and move in a relatively normal fashion. At least one cusp of the valve will become functional. However, the action will be that of a flap valve, not a flutter valve (figure 28).

In about 50 per cent of cases, at least one papillary muscle will be found to have grown directly to the valve from below. In such a case, approaching it with a straight finger (from the right side), it is quite easy to split this muscle all the way to the wall

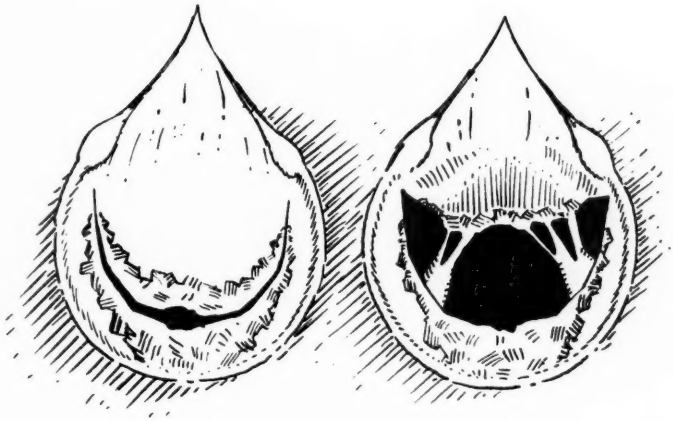

Fig. 28. The mitral valve, even although heavily calcified, can be made to open widely by extending the line of valve closure into normal tissue at each extremity of the valve.

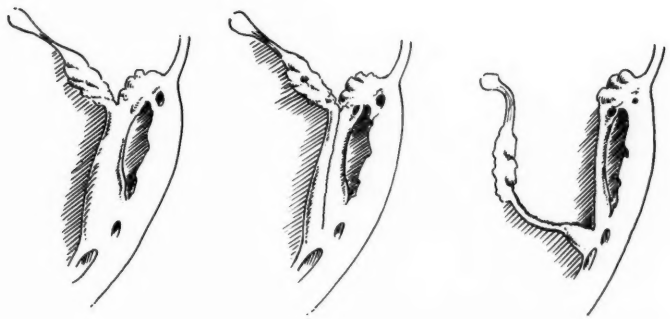

Fig. 29. Splitting the subvalvular supports of the valve when they are fused is essential for mobilization of the septal leaflet (on the left).

of the ventricle. We routinely do split such muscles all the way to the ventricular attachment (figure 29). Such splitting does not impair competence, while it does permit free mobility of the septal leaflet. Although the papillary muscle is not directly attached to the valve apex, there is significant shortening of the chordae

which may be restrictive to valve movement. Again, the appropriate treatment is splitting of the papillary muscle so that each leaflet is individually supported.

Essentially, then, this is the type of valvular mobilization that is not only recommended but the use of which is urged. We maintain that the old operation should be abandoned. It is not a very adequate procedure and recurrence of mitral stenosis[24,25] is becoming increasingly evident. We have had sixty such cases of our own, and that probably represents but a fraction of those which actually have occurred. As the years pass, more and more of them will return with this complication. But, with the new type of mobilization, mitral stenosis may be overcome completely (figure 30).

A stenotic mitral valve opened in the autopsy room shows that it opens and closes not because the rigid mural leaflet does anything, but because the flexible septal leaflet does everything (figure 31).

We have done five hundred operations using the right thoracic approach and in the last three hundred of them the new and improved technic was used. In every instance, we were able to open at least one commissure fully, and in most cases both were

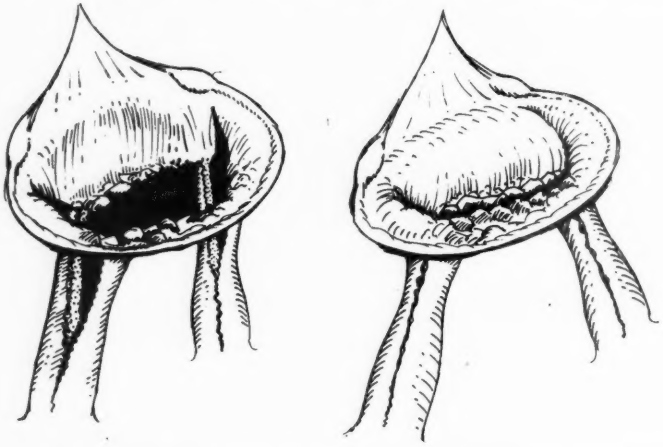

Fig. 30. A composite view of a fully opened valve, and completely separated subvalvular supports.

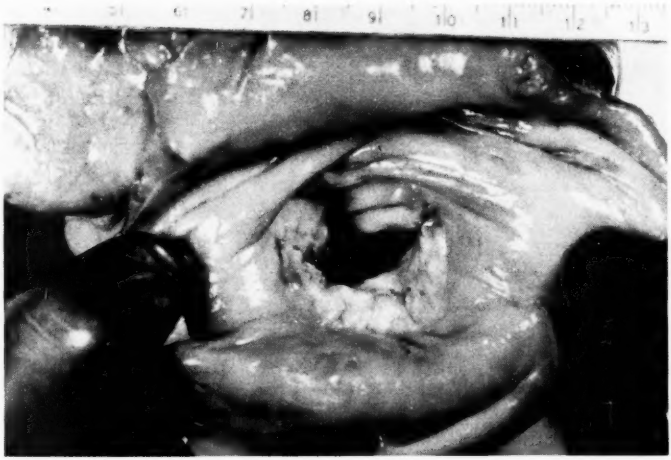

Fig. 31. Photograph of a fully opened valve, demonstrating the normal aperture which can be attained.

opened. In no case did we feel that the final valve function was less than 50 per cent of normal. With the older left-sided approach, one cannot average even 25 per cent of the normal anatomical opening, and the established per cent of function is significantly lower.

As Dr. Ellis will confirm, nearly all patients who have undergone the older type of operation have a residual diastolic murmur even after the most extensive left-sided mobilization that can be done. Half the patients operated on by the newer technic, do not present a diastolic murmur, even by phonocardiography. The other half still do. Our mortality has been half with this approach of what it was previously with the left-sided operation.

We believe that inadvertent creation of regurgitation is much less likely to occur with this technic, because one does not cut into the substance of the mural leaflet. The natural line of cleavage is followed.

Unfortunately, when surgeons first try the right-sided approach, they probably are disoriented for several operations. After that, they will begin to feel more at home. When they

have tried it a relatively few more times, the technical advantages will become obvious, and they will be glad to use it as their routine. Of course, if they abandon it after a very few cases, they never will become expert. We think that since a new type of valve is established, all the extremities of the dissection extending well into normal tissue, restenosis will be rare in the absence of a recrudescence of rheumatic reactivity. The older operation consisted on the other hand of simple division of a fibrous stricture, often at but one point. With such a technic, recurrence of the stenosis often was inevitable.

PRESIDENT GETMAN — Thank you very much, Dr. Bailey. I should like now to call on Dr. Laurence B. Ellis, whom I know will have something worth while to give us.

DR. LAURENCE B. ELLIS — As Dr. Bailey well knows, I would much rather discuss mitral stenosis than congenital heart disease, because I am at least on more familiar grounds. However, I shall confine my discussion to the congenital lesions and will bring his very lucid description of the present status of surgery for congenital heart disease into perspective, perhaps, as it relates to insurance medicine.

It is quite obvious from what Dr. Bailey said that the surgery of congenital heart disease is very complex. Depending on various lesions and developing technics, from our point of view, one can think of those lesions that are curable and hence insurable at little or no extra mortality expectation, such as patent ductus and coarctation of the aorta. It is to be hoped that the interatrial and similar defects will also prove to be really completely curable.

This will probably apply almost as well, but not completely, to the isolated stenoses of pulmonic and aortic valves. But, at the present time, of course, most surgery in congenital heart disease, with the exception of patent ductus, is done only after there is evidence of strain on the heart or a breakdown in circulation has begun to take place.

The obvious reason for this is because the surgical mortality of these people still is significant. We do not wish to subject symptomless patients to such a mortality. Hence, we are operating in a great many cases at a time when irreversible changes have

taken place in the heart and, perhaps even more importantly, in the pulmonary arterial circulation, as Dr. Bailey stressed.

One of the breakdown points in a great many of these congenital lesions, with both an increased and decreased pulmonary circulation, is the tendency to develop pulmonary vascular lesions which really determine the end of the patient medically, and may not be reversible. Until we feel that these patients can be more freely operated on before there is evidence of such strain, they will continue to be insurance hazards.

How are we going to judge cure? Of course, we have the statement of the surgeon that he has accomplished a restoration of the anatomically normal state in these mechanical lesions. His statement will be buttressed if he does this operation with an open technic, when it can be observed as well as felt. This has taken place to some extent.

Secondly, we have the changes in the clinical findings of the patient, particularly a restoration of the normal auscultatory findings in the heart and, even more importantly, completely normal electrocardiograms and x-rays, showing no evidence of residual hypertrophy. And then we have other evidence from catheterization which he mentioned.

Now, this latter method will always be of limited usefulness for insurance purposes for the simple reason that catheterization is a complicated procedure and cannot readily be applied repeatedly to patients before and after an operation. It is not only complicated, but it is also not without hazard; and we are not fully justified in catheterizing patients following surgery, particularly at the intervals when it is needed to determine fully the normal circulation.

Catheterization is also limited by the fact that the technic requires highly specialized training and the accuracy of results is in direct relationship to the experience of the catheterization team. Until newer technics are developed that are simpler, safer, and as accurate, we will still find this of limited value.

The final way, of course, of judging the value of surgery in relation to insurance statistics would be the follow-up on a large

series of patients, operated on for a sufficiently long period of time. Such a group will always be very limited in congenital lesions because they are relatively few in number and complex in nature, so that it would be virtually impossible to acquire a large enough series of more or less uniform patients.

Dr. Bailey has mentioned his own experience with interatrial defects in 150 patients, which is a very significant number. We will never have such a volume of material, nor will the insurance companies be presented with the problem such as they have had in rheumatic disease, the incidence in this disease being so much greater.

PRESIDENT GETMAN — Thank you very much, Dr. Ellis.

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PANEL DISCUSSION — PROFESSIONAL AND PUBLIC RELATIONS

ALBERT L. LARSON, M. D., *Moderator*
The Travelers Insurance Company

MODERATOR LARSON — I would like at this time to introduce our panel. Each member is also a member of our Committee on Professional and Public Relations.

Dr. William O. Purdy, Medical Director of the Equitable Life Insurance Company of Iowa.

Next, we have Dr. Charles D. Gossage, Associate Medical Director of the Confederation Life Insurance Association.

Next is Dr. Frank J. McGurl, Medical Director of the Prudential Insurance Company of America, Southwestern Office.

And finally we have Dr. Frank A. Warner, Vice President and Medical Director of the John Hancock Mutual Life Insurance Company.

What is public relations? What is professional relations? They tie in together.

We have several definitions that have been furnished us by the Public Relations Department of both the John Hancock and the Travelers. I would like to point out the dynamic aspects of public relations. It is not publicity, and it is not advertising, and it is not selling, but it touches on all of these. Indirectly, it sometimes concerns or influences the making of public opinion. All these fields may, on occasion, be helpful in building good public relations, but, basically, public and also professional relations are 90 per cent performance. What is done is performance in the public and professional interest. The remaining ten per cent is interpretation, which is simply letting the public know about the 90 per cent.

However, interpretation means very little without the primary emphasis on what is actually done; in other words, what counts is how performance coincides with the public and professional

wishes, not necessarily the general public, but, more specifically, the particular public with which a business, an organization, or an association or an individual is concerned.

With that as a background of what we are to discuss, I would also like to state clearly that we are not going to attempt in any manner to cover the entire field of professional relations in the insurance industry. We are, by definition, a committee of the life insurance group, an important part of the entire industry. As you realize, the health and accident group, sickness, and major medical groups are playing a tremendously important part in our relationships with practicing physicians. However, our task is limited to discussing our relations with our examining physicians, with attending physicians, and with the industry as a whole, as professional men and as medical directors of insurance companies.

Dr. Frank McGurl, what have you found to be the most common complaints of your examining physicians?

DR. MCGURL — In my opinion, the most common complaints concern fees and forms. Most examining physicians contend that a physical examination is the same, regardless of the type of insurance applied for, and that the time and knowledge necessary to do a good physical examination do not vary.

Consequently, they cannot understand why there is a different fee paid for physical examinations done for different types of insurance. We have had very few complaints about our fee for examinations for ordinary insurance. In a few isolated areas, where boom economic conditions exist, we have encountered small medical societies which have set a minimum fee for insurance examinations that is higher than our current fee, and have refused to do examinations for less.

In the Prudential, examining physicians' fees are paid monthly, and fees paid to other physicians for statements, etc., are paid twice monthly. I think that prompt payment of fees is an important public relations aspect of our business.

More recently, in my own office, we decided to pay the latter group immediately upon receipt of the information. Consequently, these checks are written daily, with an average lapsed time of

three days from the receipt of the information to the mailing of the check.

Most of our physicians agree that their fees are paid promptly by the home office, but, in spite of this, the examining physicians still complain. They say that the work is a drag because of delays in appointments, broken appointments, and agents' unreasonable demands for service ahead of regular patients. They complain of what they consider unnecessary requests for additional information.

The doctor feels that the applicant has given complete information on the original examination, and, when he receives a request for additional information, it implies that his original examination was not satisfactory.

It is my opinion that these complaints stem from our failure to educate the physician and our agency personnel in what is expected of them, and our reasons for such actions. In this connection, it is very desirable that we, as medical directors, establish a close liaison with our top agency people in the home office, in order to work out problems of mutual interest. In Newark, and in our various regional home offices, this friendly and cooperative relationship has helped us immeasurably in maintaining a smooth-functioning examining service.

In addition, I think, we have all experienced some change in the attitude of our examining physicians toward us in the matter of our set fee schedules. There is a growing reluctance to abide by the set fee schedule, particularly for attending physicians' statements.

As to forms for examining physicians, I think that most of us could afford to take a critical look at the various forms we use, and, where possible, combine them, simplify them, or discontinue them. In the Prudential, we have studied form revisions very carefully in an effort to simplify and to produce some uniformity of format, particularly in the shorter forms. I realize that this is a big job for all of us, but regardless of what has been done, a great deal undoubtedly remains to be done.

From time to time, various committees in our industry have sought to improve the form situation. I understand now that a

concerted effort is being made to close the gaps between overlapping committees and thus bring about a workable situation. I believe that such an effort, if implemented, will ultimately close the gap and produce the desired results.

MODERATOR LARSON — As you say fees and forms are big problems in dealing with the practicing physician.

Dr. Frank Warner, you have a large number of examiners who work with the John Hancock. I wonder how you establish your panels and about the various methods that you have used in appointing an examining physician?

DR. WARNER — We have about 7,500 examiners in the United States. We constantly revise this list and attempt to maintain as small a list as possible, consistent with giving our agents good service.

We do not object to appointing examiners if their services can be used. We emphasize that all the examiners on the panel should be used regularly. In general, I might say, in a large city, about four to six examiners per agency are about all that we feel are necessary, and usually it turns out that two are used principally, with the others as adjuncts until there is a need for their services.

We require, for the appointment of an examiner, about the same requirements that all of you have. We like to have them from grade A medical schools, with a year or two of internship. We favor the younger men. We are not frightened by career examiners, but we think that the very best way of sizing up an examiner is by observing his work. In a company such as ours it is almost impossible to do this without the help and cooperation of the underwriting departments in showing examples of good work and work that they feel is not good; and, from that point, one of our staff takes the problem and investigates it, even checking the other examiners used by that agency, to determine what the situation is in that particular area.

These examiners are routinely authorized to examine applicants for amounts up to and including $50,000 each. We have in addition a so-called special examiner list, numbering about 75 doctors. These are in the principal cities of the United States and

they are authorized to examine, on a single examination, for amounts up to and including $100,000. These examiners are rather rigidly selected. Practically all of them are board-certified internists. In addition to examining for the larger amounts, they also act as referees, and problems are referred from our other examiners to those men.

Dr. McGurl has mentioned the payment of fees. The approval of good work, and the prompt calling to their attention of unsatisfactory work, I think, are extremely important in maintaining good relations with your examiners. If you use an examiner whose work is not good, soon your examining work in that whole community will deteriorate.

MODERATOR LARSON — In other words, these examiners whom you have appointed are, in a sense, your medical department representatives on the local scene. Their qualifications and the quality of their examinations and their standing in the community reflect on the home office medical department.

How often do you visit your examiners? Do you have any set plan? And, if you do visit them, do you have any particular method of approach?

DR. WARNER — There are two answers to that. One is practical, and the other is theoretical. It is desirable, we agree, but we find it very difficult to carry out. However, we do not appear in any locality without making contact with our physicians.

I think most of you find that it is extremely difficult to visit your examiners, and we do not overlook the advantages of a good agency section to do a lot of medical relations. These men are career men, and they should be made to realize the importance of a good examination and how it reflects on the company. After all, their interest in your company is not only the commission for today, but it is the price of your product ten years from now, perhaps.

It is extremely important that medical relations also touch upon the agent. He is the one who sees your examiner every day and sometimes contacts your hospital. He may even be on the board of some of your hospitals. I think that we should rely upon our

agent. He is not our competitor, but our agency representative and our colleague in the field.

MODERATOR LARSON — When you do visit, do you have any set plan? Do you have your agency department make arrangements for a group gathering, or do you find that individual visitations are the more ideal? I suppose that can be a question, depending upon the area that you visit.

DR. WARNER — We do not have any fixed plan. You understand, we are a New England company. It is a great deal cheaper to visit the examiner in his office. You can see him operating on the ground. I am not convinced that forming a group for a social gathering is as effective as seeing the man in a small group of, maybe, one or two or three.

Most of the work, we find, will be done by two or three examiners in the area, and it is important to visit those. I mentioned the number of examiners as 7,500. Our statistics show that only about 15 to 20 per cent of those accomplish almost 90 per cent of our examining, so if we see those principal men, we will have made contact with the most important of our representatives.

MODERATOR LARSON — I think that is true in most companies.

Dr. Purdy, have you any comments to make about examiners in your company and visitations in general?

DR. PURDY — We have a little different program from that discussed by Dr. Warner. We make it a habit to visit each agency once in every five years at least. This visit is made by one of the medical directors. The purpose of the visit is to get acquainted with the examiners in that particular agency.

The medical director visits the examiner personally in his office, on most occasions; and we find that he is able to get a good look at him, see what his office setup is, become acquainted, and discuss with him any problems which he might have. It is surprising how often the examiner has questions relating to the different questions on the medical form and just what is expected of him.

If he has some complaints, we find that he is quite willing to

voice them at the time of these visits. On several occasions, we have had luncheons or dinners, but they do not produce or seem as valuable as the individual visit in the doctor's office, and I think that most of them seem to prefer that.

MODERATOR LARSON — Dr. Gossage, have you any comments to make in this regard?

DR. GOSSAGE — My experience in this matter is limited. It has not been a policy of our company to visit the medical examiners as much as, perhaps, I think, it is important to do. We have seen a few of them and the visit to the local office has been very much the more important. I believe that you get a better picture. I do not think that the doctor wants to be bothered going out to dinner particularly, or to a cocktail party, but I think that he appreciates seeing you for a brief period in his office.

MODERATOR LARSON — We have always been very well received in visiting the examiners, particularly the examiners to whom, you might say, we pay a considerable sum of money. It makes quite a difference to them and they are appreciative of your visits. In this connection there are some comments I would like to have Dr. Frank McGurl make. Undoubtedly, it is worth while for us all to hear about his system and to give serious thought to setting up, perhaps, either in a modified plan or some individual way, a similar method to that which Dr. McGurl employs. Will you discuss this, please?

DR. MCGURL — In the United States, the Prudential Insurance Company has over 15,000 examining physicians. Each of these physicians is a center of influence in his community, not only with his neighbors and friends but also in his profession. Consequently, good relations with each of these men improve our relations with the public and with the medical profession.

In my opinion, there is no substitute for personal contact with our examining physicians. No matter how much each of us would like to visit them, the problems and pressure of business in our respective home offices make such visits in areas outside our immediate vicinity almost an impossibility. On the rare occasions when we do visit other cities, we seldom have time to call

on more than a handful of these physicians. To overcome this, in our Newark home office and in each of our regional home offices in the United States, we have a layman on our staff, whose title is Medical Department Representative. This man acts in a liaison capacity between the home office medical department, our agency staff, and the examining physicians.

By reason of our continuing interest in our examining service, the medical department representative periodically visits the agency manager and discusses the over-all situation in his area. In many instances, no problems of importance exist, and those that do are resolved easily. On the other hand, if a serious problem has arisen, he will survey the entire situation and submit a report with his recommendations to the medical director.

In addition, he attends agency meetings and talks to the agents on company policy as it relates to examining physicians, what to expect and what not to expect of the examining physician and the home office, and answers the agents' questions.

I think that this is important. All of us can recall instances in which our managers or agents have been unreasonable in their demands of physicians and have assumed that they are professional employees of our companies who must do our bidding. Sometimes, the physicians' secretaries must bear the brunt of their less than civil or courteous treatment. We have a responsibility to educate our agency people to prevent this kind of thing.

In addition, our representative visits all our key examining physicians in the area. This is done solely for good public relations. If the physician has any complaints or problems relative to our work, he tries to solve them. In the case of newly appointed physicians, he answers their questions and, where possible, arranges a meeting between the physician and the local manager or other company representative.

Periodically, he supervises a quality review of the examinations of all the physicians who do the major part of our work. In addition, when indicated, he may interview some of our applicants to check the quality or completeness of the examination or the courtesy of the physician in question. Thus, while gathering

facts for us, he creates good will. Many physicians have commented very favorably on this policy, and look forward to his visits.

MODERATOR LARSON — It seems that you have a very interesting situation here, where you have a layman who makes periodic visits to physicians. Have you had any reaction on the part of the physicians to having a layman come around and visit them and discuss these problems with them?

DR. MCGURL — The reaction, in my experience, has been very favorable. We have received any number of letters from physicians and personal calls from time to time, indicating that they not only enjoy his visit but look forward to having him in their office.

MODERATOR LARSON — Of course, that is directly related to his qualifications and training. What, briefly, do you require in an individual who is a medical department representative?

DR. MCGURL — The man in my office typifies any of the men in our respective offices. This particular man is a college-trained man, a college graduate. He is not fresh out of college, because we desire some maturity. Consequently, he may be between 25 and 30 years of age when he starts this job. He is a very personable individual, has a good personality, a very nice appearance, cleancut, trim. He meets people and physicians very well. He need not have any particular training in medical background or in underwriting or anything of that kind, but, when he comes to us, we give him training, of course, usually by sending him to Newark for a period of six to eight weeks.

He works right in our medical department in Newark, and becomes familiar with our forms, procedures, policy, and our philosophy. After that he goes out for a period of four to six or eight weeks with one of our medical department representatives who has been doing this work for years. He works closely with him in the field. After that, of course, he comes back to our regional office and carries on from there.

MODERATOR LARSON — What do you think of this system, Dr. Warner?

DR. WARNER — I think that it is an excellent system, and has a number of advantages. It does not require a highly paid medical director to be away from the home office. Many of us are short-staffed. If you can get a layman and train him, I think it is an excellent system.

There is a possibility that some of us follow a plan not unlike this in some respects, though, perhaps, not so highly developed. We have, on occasion, used what is called our Bureau of Investigation, because they know the physicians in many of these communities; and in a special situation, we may direct one of the representatives to visit the physician, or one or a series of persons who have been examined.

We also have a young man, a layman, in the home office who works with our group department, and he and my department work very closely together. He goes out occasionally to see physicians. He does that after talking over the problems, and sometimes he goes as my representative. But we do not have a system as highly developed as Dr. McGurl's.

MODERATOR LARSON — I think that we have covered the various methods of contacting, appointing and handling our examining physicians. We would all agree, I believe, that it is our medical department's reflection in the field. Economically, perhaps we cannot all have the same type of system as the Prudential has developed. Yet, on the other hand, perhaps it is more convenient than trying to have the medical department send physicians out to the field.

I am sure, if there is trouble the medical man himself goes out and personally consults the physician. But there is an important channel in that the chain of command is directly into the medical department. There is not another agency group or underwriting group intervening in medical matters, and this is a very important thing to consider in our relationships with physicians. Too many third party individuals may sometimes spoil the rapport.

DR. GOSSAGE — Mr. Chairman, I think that this contact through a lay medical officer sounds excellent and would appear to be working very well in this country. I would just mention that possibly, with us, there might at first be some resentment to a

lay contact. It is wrong that there should be, perhaps, but it is vitally important because the whole success lies in the individual and the choice of the individual.

I think it would be a very bad start if you should have, by any misfortune, a second rate representative. The fact that he is a layman would spoil it at once.

MODERATOR LARSON — A sort of medical department detailman, you might say, is what we have described here. Dr. Purdy, you have done a lot of work in analyzing statistical statements of attending physicians. I wonder if you would mind discussing, in general, a recent very well thought out and constructed analysis of attending physicians' statements and their worth to you in underwriting?

DR. PURDY — First, I would like to say that we were prompted in making this study because of the concern on the part of the personal physician in having so many inquiries from insurance companies.

The second thing that prompted us to make this study was the cost of securing reports from personal physicians.

The third thing that caused us to become interested was the matter of delays in the issuance of business, pending the receipt of these reports.

The procedure was to poll all the cases to which we had addressed a personal physician's inquiry for the year 1955. These cases were reviewed by one of the medical directors, and the pertinent data recorded on IBM cards, so that we could obtain almost any of the information desired, regarding sex, age, or anything else.

We found that during 1955, we had written to 3,292 personal physicians. I will not go into detail about the results of this study other than to mention the things in which we are interested for this discussion — the value of the reports which were received, both on the medically examined and nonmedical cases; the percentage of cases on which inquiries were made; the time required to secure these reports; and whether an inquiry made

solely because of a declaration of a check-up examination produced significant information.

We found that we had required reports on 17 per cent of the cases submitted during that year, at an average cost of $3.15. The reports were received in the following order:

7 days or less	—	39 per cent
8 to 14 days	—	31 per cent
15 to 28 days	—	23 per cent
over 28 days	—	7 per cent

On medically examined cases, 70 per cent of the reports permitted standard issue; 14 per cent were responsible for the case being rated; 3 per cent made declination necessary; and 12 per cent improved the case.

In the nonmedical group, the findings were quite similar, or practically the same.

When we analyzed the medically examined cases on which an inquiry was made solely because of a history of a physical check-up, we found that 88 per cent of the inquiries permitted standard issue; 12 per cent were responsible for the case being rated; and 2 per cent made declination necessary.

On the nonmedical group of cases, 95 per cent of the reports permitted standard issue; 4.5 per cent were responsible for the case being rated; and 0.5 per cent made declination necessary.

We concluded from this study that the reports received from the personal physicians were very valuable in underwriting. We also concluded that we could restrict quite materially the requests for information on declarations of check-up examinations in the nonmedical group. There, we were dealing with a younger age group and smaller amounts, and the cost of securing the report being quite considerable, we felt that we could reduce the number of requests. I think that was what we had hoped to do — to find some means of taking some of the load off the personal physician in replying to so many inquiries.

As far as the time element is concerned, we felt that the physicians were giving us fine service.

MODERATOR LARSON — You also made another survey, taking a sample of a group of companies. I wonder if you would tell us why you did this, and also the results of that study.

DR. PURDY — The reason why we did it was because we were contemplating making some change in the handling of our inquiries and I might say that this survey was conducted through 46 life companies, including our own.

We found that the request for information from the personal physician was handled in different ways. Seven companies secured the information from the personal physician by means of a personal letter over a medical director's signature; 12 companies used a combination system of a personal letter or form; and 27 companies employed the use of forms for this purpose.

We also included a question regarding fees and, of the 46 companies surveyed, found that about half or 22 companies sent a fee with their letter of inquiry or form as a routine procedure. One company indicated that they had employed this method for a considerable period of time and it had proved unsatisfactory for them, and they were going to have to discontinue it.

MODERATOR LARSON — This question of sending a stated fee with the request for the attending physician's statement, in one case, did produce a reaction that made them stop. However, here you have a group of 46 companies surveyed and approximately 50 per cent of them were sending a fee directly to the attending physician when making the original request. Would you have any comment to make on that system, Dr. Gossage?

DR. GOSSAGE — We send the fee with the request and the fee is based on the minimum fee required in the local schedule of fees published by the local medical association. We do not offer less than the fee stated there, and we say that "We enclose herewith the usual correspondence fee. We trust this will be satisfactory. If it is not, please enclose your account." That may be wrong, but we have rarely had an extra account enclosed. I think that it removes that element of dictating fees which is very bad.

I may say that when we look up the rating of each physician written to, if he is a certified specialist, as we would call him, we

offer five dollars; if he is a general practitioner, we offer him three dollars. That seems to be a way of avoiding some of this trouble.

MODERATOR LARSON — Do you have any comments to make on this, Dr. Warner?

DR. WARNER — We do not use the prepayment system because it has not been our feeling that doctors doubted that they were going to be paid. We feel that the system has the disadvantage of suggesting to the doctor that this is the fee for this statement.

We are making a study between two sides of our house, the one in which we ask the doctor to name his fee, and the other side in which we have set three dollars for this statement. We do not know the results as yet. I speak from impressions on this matter.

We have no hesitation in paying a doctor five dollars for his attending physician's statement. If he wants much more, he has to prove it in the quality of the statement that he sends. We have not felt the need for enclosing the fee.

MODERATOR LARSON — We do not use that method. As a matter of fact, we write directly to the attending physician from the home office. A personal letter goes to him, accompanied by a blank sheet of paper. We do not use any form. If he wants to use his own stationery, all right — otherwise, we give him a piece of paper to write on.

We ask that he submit an account for this service. We have been doing this for a number of years. The average fee paid an attending physician is interesting because in the last 7,000 cases that we considered — that is part of this year — the cost was $3.57 per attending physician's statement. Of course, this includes many that cost five dollars, and the number is increasing to whom we are paying five dollars.

Our procedure at the present time is to state in our letter, "If you are a member, for example, of the American Board of Internal Medicine, we will pay for your report as an internist at an increased rate." We are not discussing accident and health insurance, but it just so happens that in the last 800 inquiries of attending physicians relating to the underwriting of accident and health, the average cost was $3.32.

Does that coincide with some figures that you have, Dr. McGurl?

DR. MCGURL — Approximately a year and a half ago, my own office and the office in Minneapolis made a study over a five week period on this subject, on various aspects of the attending physician's statement.

We sent these statement requests out on a special form in which no fee was stated, and asked the attending physician to insert his own fee or return the completed form with the bill. At the conclusion of this five week period, the number, as I recall it, was approximately 900 for both offices.

When a doctor did not submit any fee, we automatically credited him with three dollars, and the others we paid whatever they asked. Dr. Schaefer in Minneapolis had the unfortunate experience of having one doctor request a fee of twenty-five dollars for what he thought was possibly worth five dollars. It threw his statistics out of kilter, and he wrote to this doctor and told him it was just a study and, perhaps, in fair play, he would reconsider his fee which he did not.

The outcome of this study, as I recall, was that the over-all average was somewhere between $3.25 and $3.50; about $3.27 or $3.30, as I recall it, per attending physician's statement.

MODERATOR LARSON — That pretty much coincides with much of our experience. The highest one I have seen recently was a fifty dollar fee for a statement. We are not particularly discriminating against psychiatrists, but this happened to be a psychiatrist who wanted fifty dollars, so we interpreted that he had misplaced some decimal points and sent him five dollars. That did not satisfy him at all.

Dr. Purdy, we all borrow a lot of essential material from the files of the physician for underwriting cases. These are the electrocardiograms and x-rays, and the meticulous handling of this material is extremely important. In our grievance file, we have noted this to be one of the major points that physicians comment about in their relations with insurance companies. Have you a system for handling that?

DR. PURDY — Yes, we have a system. The mailing section will send any material of that type, electrocardiograms or x-rays, direct to one medical secretary. It is her responsibility to make a record of those special studies so that after we are through with this material she can return it to the proper source. When she returns these studies to the doctor or to the hospital, she writes a personal letter, thanking them for the use of the material, and enclosing a check for whatever fee has been stipulated or required. We try to see that these materials are handled so they are not damaged. We put them in fresh envelopes when they are returned, and so on. We think that is very important for nothing disturbs a physician more than to have his material lost, misplaced, or delayed in getting back after he has lent it to someone for review.

MODERATOR LARSON — Do you request this just in specific cases, or do you have a rule for an amount basis or anything like that?

DR. PURDY — We have no fixed program. We are using the electrocardiogram and chest x-ray frequently in our work, and occasionally encounter changes in a tracing where there may be some question of the significance, and it is always nice to be able to get an old tracing and make a comparison. The same thing holds true for the chest film. We request these studies very frequently. I think that we are doing much more of this type of thing than we did ten or fifteen years ago.

MODERATOR LARSON — Dr. Warner, do you have a method?

DR. WARNER — I think that ours is roughly similar to that described by Dr. Purdy. I would like to stress, too, that it is important that these studies get back to their owners. We have tried to make ours automatic, so that the studies go back with great promptness. We have one girl assigned to this. As you know, physicians are different in their manner of submitting these reports. Some materials are loose and may be left in the envelope if you have someone in the office who is careless in opening these. So these envelopes as they are received in the office are all opened by one person, and she is trained to look for those things, everything that the doctor has sent in, and also

to be responsible for them getting back to the attending physician.

MODERATOR LARSON — This brings us to another very important point that has been stressed in these meetings for some time, that is, the confidential nature of the attending physician's statement. Dr. Gossage, perhaps, you might say a word about how that should be handled?

DR. GOSSAGE — It is most important that this should remain confidential. Of course, our whole staff in the underwriting department is supposed to be in a confidential position. Very confidential information should be kept by the medical director alone. Sometimes, we get some of that, but not very often.

If we receive some adverse information, we should attempt to develop that information ourselves rather than use what we have been told. We also respect the wishes of a physician who does not wish something revealed to his patient. That presents a rather difficult situation, where somebody, perhaps, has a malignancy of which he has not been told. I think that one must exercise complete confidence there. It is difficult, but it can be done.

If we do not respect the confidential nature of things, we will lose the right to receive them.

MODERATOR LARSON — I certainly agree with you. This particular subject, as I said, has been discussed here before, and, looking through the files of the committee regarding grievances, it is one of the most important things written about by attending physicians. If we do not carefully control this information, our sources will be drying up gradually, and there has been some impression that some of the most important sources of underwriting information appears to be disappearing slowly because of the loose handling of very pertinent confidential information.

It seems to me that there is no question but that we should, in practically all cases, not in every case because no rule can be made 100 per cent effective, do as Dr. Gossage suggested — go back and try to develop, through directed reinterrogation, the material that is necessary for the underwriting of the case. Dr. Warner, do you agree?

DR. WARNER — There is no doubt about it. The question here is, how much do you tell your agent? Your agent is presented with the difficulty of submitting this rated case to the applicant. You have to tell him something; otherwise, your agent is going to look just a little ridiculous if he cannot tell him.

Our system has a number of weaknesses, because, I think, we have overemphasized keeping information confidential. It has not been our habit to tell agents much. We will tell them for simple impairments such as elevated blood pressure, changes in the urine specimen, but, if it is anything else, it generally goes out as "this action is based on medical reasons". We have no objection to furnishing it to his attending physician but I think that we are placing our representatives at a disadvantage. Do you inform your agents?

MODERATOR LARSON — We inform them in a general way, through our underwriting department. As a matter of fact, if I rate a case or decline one, I indicate in a certain area on the file the reasons for doing so, and those reasons are couched in general terms. We tell them that we rated them because of elevated blood pressure. We might use the expression, "variable blood pressure findings".

We pick up, as you all know, many important physical impairments during the course of a life insurance examination — glycosuria, heart murmur, elevated blood pressure, and hematuria. I do not know how many people have been examined by the cystoscope because the urine specimen sent to the home office contained red blood cells. Urologists should be our good friends. Nevertheless, if we discover such things we tell it in general terms to our agency department. We do not feel that we are trying to practice medicine but add a little note that our applicant would be well advised if he would consult his personal physician, to whom we would be willing to give pertinent information upon written authorization from the applicant.

Is that about the way you handle these, Dr. McGurl?

DR. MCGURL — Yes, that is essentially the way we handle it. We feel very strongly that any information supplied to us from confidential sources should be kept confidential. Consequently,

any information that comes to us that is omitted by the applicant, the medical history or findings on the physical examination, is communicated to the agency in very general terms. But, anything that comes to us through inspection sources, to which the applicant has not admitted, or anything from an attending physician or a hospital, to which the applicant has not admitted and we have not developed on physical examination, is kept confidential. In notifying our field people, we have a code of numbers and we simply indicate that that is confidential. We do not relinquish that information to anybody.

MODERATOR LARSON — That is a very good point. Dr. Gossage, you probably have been a more recent practicing physician than any of us here. I wonder if you would discuss, in general, how we as professional individuals and medical directors may enhance the respect of our colleagues in practice.

DR. GOSSAGE — Our public relations are very largely dependent upon the regard which our clinical colleagues have for us, first, as professional individuals, and, secondly, as the medical directors of insurance companies. I think we are known by our deeds.

First, as professional individuals, we must certainly be well and favorably known by our clinical colleagues, both for ourselves and for our professional ability and judgment. We cannot be known and we cannot retain our professional ability unless we retain contact with clinical medicine and our clinical colleagues.

There are several methods by which we can do this. I am sure that these are all well known to you, but, perhaps, repetition will not hurt. The first method is by retaining an appointment in a clinic, in a hospital, in a teaching center or some such thing. Companies vary tremendously, of course, in their attitude toward allowing their medical directors to work in clinics. I know of one very prominent firm which will not retain a medical officer unless he has a teaching appointment in the local university. They retain him full time, and send him to the university half time for teaching and clinical purposes. That is, perhaps, one extreme. There are few other companies that will go to that extent. But, generally speaking, companies will make one of their men available for some period of time to maintain a clinical contact.

I should think that this would be very important where we retain younger men in the insurance industry full time, before they have established themselves as thoroughly as they might in their profession and where they still need to continue furthering their clinical information. That is one phase.

We should make it possible for our colleagues in the insurance medical industry to attend refresher courses occasionally. There, they mix with their colleagues who realize that they are in the field of insurance medicine, and are still keeping abreast of clinical medicine, and they admire them for it. How often that can be done depends, of course, on necessity. Sometimes, about every five years, a person might reasonably get away for a few days or a week for a refresher course. I think that it is very important.

We might well be able to keep attracting desirable young men to the field of insurance medicine if they realized that we were interested in keeping them clinically alert and allowing that sort of thing.

The second important method of keeping abreast and in touch, of course, is through attendance at meetings of your local clinical society, your federal or your provincial or state medical association. This is very important, and some of our group are undoubtedly capable of giving very useful papers at these, and they should make a point of doing so. If we are not able to give a paper of clinical interest, perhaps, then surely we can take part in the discussion and bring to that discussion the invaluable help of the long term prognostic value of our insurance statistics.

There is also, from time to time, especially in these days with increasing socialization of medicine and prepaid plans, not infrequently a demand for a medical director to join a panel discussion at the local academy to present the insurance point of view. He should be prepared, willing, and able to do so.

There are also many executive jobs in the local associations that we should willingly take on.

Third, we should not forget our scientific responsibilities as physicians, as medical directors, and as presumed leaders in the community. We should be willing and prepared to accept our

civic responsibilities, and that might well be done in sort of ancillary medical situations, such as taking executive work in the Red Cross. There is an increasing tendency for the professions to assume the responsibility for their own canvassing in Community Chest work. I think that most of them feel it is important that the chairman of that committee be, perhaps, a clinical man from one of the hospital staffs. But, the deputy has a great deal to do, and he can well be an insurance medical director, because usually his company will make him available, give him the time, secretarial assistance, and so on, and he can be the "worker boy" of that team. This is much appreciated by the local medical profession and has been done, certainly, in our own locality. In fact, we have gone so far as to suggest, and so far have been able to do so, that we will find that deputy among the various members of our group.

Therefore, by these various methods, we can keep ourselves *persona grata* with our clinical colleagues, and that is essential, as well as maintain our professional ability.

MODERATOR LARSON — You also have some connections with committee work, subcommittee work, or study groups, as you might call them, where, actually, you discuss with practicing physicians and organized medicine in Canada certain aspects of insurance matters. Would you tell us about that briefly?

DR. GOSSAGE — We had an interesting situation. Our situation, of course, is a little different from yours in the United States. Our population is not so large. Perhaps, we are in closer contact with our opposite numbers than you can be. We have a body known as the Canadian Life Insurance Officers Association, which is somewhat akin to the American Life Convention, and is a group of top executive officers of our Canadian insurance industry. They have many committees, of course, and one is a standing committee on accident and sickness insurance. There is a subcommittee of that known as the Subcommittee on Relations with the Medical Profession.

There was a most extraordinary situation there in that there were no medical representatives on this committee. They suddenly became aware of this, and requested the Public Health Committee

of the Canadian Life Insurance Medical Officers Association to nominate three men to it, so three medical officers were nominated to it and have been a part of that committee since, I think they have been able to bring some value to that committee.

One of the first things that was accomplished was to point out a good many irritations to the medical profession and that we should do our best to get our companies to remove some of them. As a result, we were asked to bring in a sort of sub-subcommittee report, which we did, dealing with such matters as the importance of having requests for medical information signed, at least by the facsimile of the medical director's signature, rather than a layman, and so on, avoiding the dictation of fees and many of these things which have been discussed.

That report, which covered some six or seven items, was accepted by this committee. It was further accepted by the executive, and finally circulated to the member companies, to the presidents of the member companies, and finally came down through that channel to the medical and underwriting departments. I assure you that information and suggestions coming via that route seem to have much more influence than anything I might do if I go home, filled with enthusiasm from this meeting, with a lot of good ideas, and try to put them across by myself. A prophet is not without honor save in his own country, and though I am sure they hold us in some considerable respect, nevertheless, it does help when it is a considered report coming through, as it were, trade channels.

A further development of that committee has been a study group. We have many mutual problems with our professional colleagues, and the executives of our provincial and federal associations are well aware of them and most anxious to do something about them.

We approached them and, without any difficulty at all, arranged to have an informal study or discussion group with their executives. This was done in a very delightful and charming way. We had a very good dinner, with all the usual trimmings, which did help, and we started a discussion group which has continued. There have been three dinners. We have no terms of

reference. We have no agenda or anything particular to deal with. It is simply an informal discussion group to air problems and hear each other's point of view.

Through that means, there has been established an excellent rapport. There is no doubt that when we have a problem with which to deal specifically, we will be able to get an official committee appointed and ready to go to work on it.

Also arising out of that was a direct invitation from the *Canadian Medical Association Journal* to edit a page or a column, or as much as we wanted, on insurance medicine, or matters dealing with insurance medicine. We hope to undertake that. Of course, that will be done under the name of the Canadian Life Insurance Medical Directors Association. There will be no person or company mentioned and credit will be given for anything we abstract or reproduce.

By those means, we feel that we have accomplished something. We have suggested that they devote a period of time, perhaps a half day or at least a few hours, to the discussion of insurance problems. I think that may happen. This, I believe, is a very worth while liaison.

Earlier in our meeting, it was said that the Health Insurance Council here was establishing state committees, and it made me think that, perhaps, we had "beaten the gun" a little, for we already have a committee which can develop into anything that might be desirable.

MODERATOR LARSON — Thank you, Dr. Gossage. That was a very valuable comment. Have you any comments that you would like to add to this, Dr. Warner?

DR. WARNER — I think that Dr. Gossage has a fine program and he has described to us just what can be done if we interest ourselves in this problem. It takes a lot of extra work and it may mean that you will take a briefcase home at night. That requires a great deal of tolerance on the part of your family, but if skillfully handled, it can be done.

Dr. Gossage did not mention the Board of Life Insurance Medicine, though I am sure he meant to do so. Just for the sake

of completeness, may I add that? I think that is very important, to continue with our staffs to inform themselves on the insurance operation within their own companies and outside their companies. There are some companies, I think, that do not subscribe to the view that we should continue our clinical interests.

Several of my staff enjoy important connections with local hospitals. It is a voluntary status. It is not a requirement in my company that we spend half our time teaching in a university, but that fits the situation in the locality that was mentioned. I think that it is a great program and a credit to the life insurance industry that we have men such as Dr. Gossage, who show us what can be done if we interest ourselves in it.

DR. GOSSAGE — May I hasten to add that I did not say I was on these committees or that I did these things. My colleagues have worked very diligently with us.

I do want to say that I regret not having mentioned the Board of Life Insurance Medicine, to which I pay great tribute. I have attended all of their courses and think they are excellent. I hope they continue. I thought we all took them for granted as being thoroughly established and essential to our work.

MODERATOR LARSON — I am certainly glad that we can pay tribute to the Board of Life Insurance Medicine. It deals with education and professional aspects of our business.

I heard a very good comment yesterday from one of the prominent members of our organization, in which he stated that a postgraduate education lasts about five years. If you do not renew it about every five years, it loses its values, just about the same as the value of an insurance examination, the select period. It is important to all of us at times to renew our interest and acquaintance at least every five years, and the Board of Life Insurance Medicine and the program that was outlined by Dr. Pepper on the first day of the meeting will help to do that.

Gentlemen, thank you very much for your efforts in this panel. We have covered, in the time allotted, the aspects of life insurance where we deal with the practicing physicians who are our examiners, who are the attending physicians, and also our relations

between the profession and the medical directors. Of course, we could not make any recommendations to you, but we do gently urge you on returning home, to take a critical look at some of the practices which could, perhaps, be eliminated and become less of an irritant to our associates. Thank you very much.

PRESIDENT GETMAN — Thank you, each and every member of the panel. They told me this morning that they were not going to offer anything new. I should say that they had offered some things that we should all keep in mind at all times. They certainly have polished them up and made them sound new, if they have not been.

Our second panel this morning will discuss the "Economics of Health Care". I am sure that you will be interested in the contributions of each of the distinguished members of this panel. The Moderator will be Dr. Howard L. Hauge, Medical Director of the New York Life Insurance Company and a member of the Accident and Health Committee of this Association.

MODERATOR HAUGE — This part of the program has been listed as a panel. Actually, it is not a panel in the true sense of the word. We have, however, assembled for you three experts who are going to discuss the economics of health care. They will give short talks on the various aspects of this problem.

The gentlemen who will speak to you are well qualified to discuss the various aspects of the problem. Questions from the floor will be entertained, but I ask that you please hold your questions until all the talks have been completed.

Mr. J. Henry Smith, Vice President of The Equitable Life Assurance Society, and President of the Health Insurance Association of America, will speak first. He is an actuary. He will discuss this problem from the standpoint of costs, premium rates, and the interdependence of those costs and premium rates on the attitude of the practicing physician.

The second speaker will be Dr. Percy E. Hopkins, Chairman of the Committee on Prepayment Medical and Hospital Service of the American Medical Association. He will discuss the problem from the point of view of the practicing physicians, some of

their complaints, as well as an analysis of their responsibilities in this field.

The closing speaker will be Mr. Albert V. Whitehall, Vice Chairman of the Health Insurance Council. He will speak on the health insurance partnership and the interdependence of the Health Insurance Council and health insurance.

Mr. Smith, our first speaker, has had a wide experience in this field. He served as Chairman of the Health Insurance Council from 1949 to 1950; Chairman of the Joint Group Life Insurance Committee of the Life Insurance Association of America and the American Life Convention from 1952 to 1953; and as a member of the Board of Governors of the Bureau of Accident and Health Underwriters from 1952 to 1956. It is my pleasure to introduce Mr. Smith.

THE "ACHILLES' HEEL" OF HEALTH INSURANCE

J. Henry Smith

Vice President and Executive Assistant, Equitable Life Assurance Society

President, Health Insurance Association of America

I never face a group of doctors without some awe and without a certain degree of envy. It seems to me that your profession must be among the most fascinating of human pursuits. To study and be concerned daily with the marvels of the human machine could hardly be surpassed in interest! Furthermore, ennobling concepts of service to one's fellowman, which is the whole purpose of the physician's activity, must provide about as soul-satisfying a career as one could find.

I am glad to find, however, some of the same elements in the business of health insurance. In many respects its social impact and implications are like, in fact are closely interwoven with, those of the medical profession. And it, too, is a fascinating field. For example, to the salesman, health insurance is fascinating not only because it finds a ready market and yields good commissions, but he learns that he can properly serve the market only if he exercises due concern for the needs of his clients and if he looks on his job as a professional counselor in a field that is likely to bewilder his clients. The adaptation of the many ideas current in health insurance to the needs of the public, calls for skill, devotion and a high order of service.

The insurance administrator finds the health coverages absorbing because they are complex and because they have grown at such a fast pace as to continue to challenge him in his organizing and planning.

The actuary is intrigued because health insurance presents technical and economic problems of a complex order. He is used to coping with probabilities, but in this line he must also allow for important subjective aspects of health, for possible rapid economic changes and for the attitude of people not actually parties to the

insurance contract, for example, doctors, and hospital administrators.

The physician, also, is involved with health insurance to such a degree and in such important respects as to make it a significant subject for him. The practitioner, on the other hand, is likely to find that his income is derived in large part through insurance nowadays, and in turn he should realize that his attitudes, habits and decisions vitally affect insurance. On the other hand, the insurance company's medical director will find this a compelling field. He can be most important in it as an advisor and interpreter.

As you may suspect, the purpose of my talk is to elaborate on this theme of the involvement of the doctor in health insurance. Before going on with it, however, let us take a brief look at some insurance and actuarial fundamentals. The success of any kind of insurance depends on reasonably accurate, or at least conservative, predictions of the risk involved in the contingency to be covered. For life insurance, we have to know something about how many people are going to die out of a given group with certain characteristics. For fire insurance, it is the rate of burning in combination with the average cost of replacement. And so on through all the different forms. Now, for health insurance, however, we find that there are several elements to consider and that some of them are on the indeterminate side. Let us pretend for a minute that we are actuaries trying to find the right price for a particular health policy. The elements we have to take into account may be catalogued as follows:

1. The *definition* of the contingency to be covered.
2. The *frequency* with which the contingency occurs in the insured group.
3. The *benefits* to be provided when the contingency does occur.
4. Amount needed for taxes, for expenses in selling and operating the insurance, and for a safety margin.

The first of these, the contingency to be covered, sounds simple at first, but actually in health insurance it usually presents a complex problem in definition. Ill health is an entire spectrum grading continuously from the imaginary to the vital. Exactly what part of that spectrum will this health policy cover? Will it be a single

cause like poliomyelitis or the whole range of causes? And what services are to be covered — those of the doctor, nurse, hospital, podiatrist, druggist, diagnostician, or laboratory? Today the trend is toward comprehensiveness in policies; but exactly how is the scope of this policy to be defined so that there can be some precision in its administration as well as in the estimates which the actuary must make?

Well, for our simple illustration, let us assume that we are trying to find the price of a policy covering only the fairly simple event of confinement to a hospital. This sounds easy to define and administer, and it is easy compared to some kinds of health insurance. But wait a second — what do we mean by "hospital"? Do we cover nursing or convalescent homes, places for the chronically ill, the office of the doctor who keeps a patient overnight when he removes tonsils? How about the mental institutions, and the long list of municipal, county, state and federal institutions, including veterans' facilities where care is often free? Amazingly enough, there is also the demand that we cover Christian Science hospitals!

Over the years now we have learned how to handle the definition of hospitalization for insurance purposes, but you can imagine how complex and uncertain are the problems of definition when we begin to broaden out toward really comprehensive medical care. On the other hand, maybe we could simplify the problem if we could write a single completely comprehensive policy to cover every conceivable health cost. We have made important steps in that direction (particularly under Major Medical type policies) and maybe we can go all the way some day. But, from what I hear about the kinds of things that are called medical costs for income tax purposes, I wonder. They tell me some businessmen deduct the cost of Turkish Baths and gymnasium work-outs! So, for the present we still have to worry about definition of our coverage.

But to get back to our simple actuarial problem, we are going to cover hospitalization, and nowadays we have a good working definition of it. Now, for the second step — what is the frequency to be expected? Fortunately today there are lots of statistics based on past experience and catalogued for different kinds of hospitals and characteristics of insured persons. If we can be sure that past experience is a good guide, we can evaluate this item closely.

Let us say that for the plan we are going to write, statistics suggest a rate of ten confinements per year per 100 persons covered.

Now the next step — what benefits are to be paid? Here again we have some decisions and definitions to make. What hospital charges are we going to recognize? What durations of hospitalization will be covered? How do we take into account the various ways that hospitals use in allocating and billing their costs? What do we do about the medical services performed by doctors on the salaried staff of some hospitals? You can imagine a half-dozen more questions.

Let us decide that we will provide for semi-private room and board for a maximum of 70 days, and that we will cover all other charges made by the hospital up to $300 for any one confinement. Examining our statistics, suppose we find for our plan that —

The average stay (limited by the 70 day maximum) is eight days.

The average semi-private room and board charge is $15 a day.

The average of other charges (limited by the $300 maximum) is $120 per confinement.

The combination of these figures is —

$8 \times 15 + 120 = \$240$ benefit per claim. (Do not take these figures literally — they are illustrative only.)

Now suppose there are ten hospitalizations per year per 100 at an average cost of $240. This is a total of $24 per year per person which we must have to pay claims. In addition, we need expenses, reserves and margins, which our experience permits us to estimate fairly readily. Maybe we need $6 for this, making the total required as a premium $30 a year per person. Simple, is it not? I do not know why people think actuarial work is mysteriously complicated.

Of course, other forms of health insurance are more complicated to compute. Furthermore, in many forms we do not have much in the way of statistics to guide us. We have to make "educated guesses" as to frequencies, durations and average costs.

What is more, even for the standard product of hospitalization insurance, certain factors may come into play not contemplated in

our statistics. For example, after we underwrite the insurance, suppose more people are sent to the hospital than used to go. Suppose that instead of ten per 100, the actual frequency turns out to be 11 per 100, because of changes in medical practice or because the existence of the insurance takes the financial brakes off of, or even encourages, going to the hospital. Then perhaps our claims will run over $2,600 per 100 instead of $2,400.

It is also possible that the average stay in the hospital will increase. We have been lucky in the past in that average durations of hospitalization have gone down, but the figure seems to have hit bottom lately and may bounce a bit. It often seems to bounce when insurance takes care of the cost. In our example, an increase of one day in the average increases the cost at least $1.50 a year for each insured person.

Even more likely, the hospital's charges for its services and supplies may go up. New medical technics and drugs, higher salaries and other costs, and the tendency to "shoot the works" at the cost of the insurer may push our average claim up many dollars. Experience lately has been just that. Maybe in our example, the semi-private room charge goes up 5 per cent — as it has about every year lately — and under our $300 limit actual payments for ancillary charges average $130 instead of $120 expected. Let us see where we stand now on our hypothetical premium of $30 per year per person —

Suppose the frequency is 11 per year per 100, instead of ten.

Suppose the average duration is 8½ days per confinement, instead of eight.

Suppose the average semi-private room charge is $15.75, instead of $15.

Suppose the average ancillary charge is $130 per case, instead of $120.

Not one of these changes seems to amount to much, nor to be beyond what we might expect. Let us see, however, how the actual cost then turns out to be in comparison with our $30 premium:

$$11\ [15.75 \times 8\tfrac{1}{2} + 130] = \$2{,}902.68 \text{ per } 100$$

or $29.03 per person. If we are collecting $30, we have $0.97 left

for expenses, whereas we figured we needed $6. There is a $5.03 shortage on each person.

You may say, "Well, that is hypothetical!" Yes, but it is also representative of just what is happening today in all too many situations. And it is illustrative of why we must worry about trends and attitudes.

It must be pointed out at once that much of this cost increase is quite beyond the control of doctors, hospitals, insured persons and companies. Changing price levels is considerably responsible. For some forms of insurance, most notably Major Medical, where benefits depend directly on prices without much restraint, the effect of inflation can be of extreme importance. We need the Major Medical forms for social reasons, but if price levels rise sharply, these forms may prove disastrous. Therefore, health insurance companies and their friends have every reason to do everything they can to arouse the country against the evils of inflation. We are glad to see evidence of rising popular concern in this matter.

In the recent past, medical costs have had a special inflation all their own, going beyond that of the economy generally. This has been due, in large part, to the economics of hospitals which have changed from those of a benevolent institution into those of an expensive service business. It is also due in no small part to changes in medical technics and the development of expensive drugs, procedures and apparatus. Further changes along these lines tending to raise the cost of health will create a difficult set of problems for health insurance of the same nature as general inflation would produce. Here we feel somewhat powerless to prevent the effect, because no one wants to restrict improvements in medical technics however expensive they may turn out to be, although we are hopeful that they may be offset in cost by economical practices in the handling of patients and by effective disposition of medical personnel and facilities. Such moves as an early shift to outpatient care and inexpensive domiciliary care of the chronically ill and aged, may prove of real help to health insurance in offsetting other expensive trends.

In addition to the perils of inflation, however, note in our hypothetical case that the future of insurance also depends on the

attitudes of those engaged in the health professions, particularly of the physicians. Let me illustrate this further with some practical examples. What are some of the attitudes that can hurt importantly?

I will try to make some of these emphatic by a few rather outrageous analogies. For example, a few months after one of the hurricanes hit our town about 1954, a tree surgeon called on me and pointed out that all of the several trees on my property needed pruning and expert care. He quoted a figure that I thought exorbitant, but then he quickly explained that he would be glad to certify that the work was required because of damage resulting from the hurricane. This would permit me to claim windstorm damage under my insurance policy. Here was a way, a device, for him to make a high fee and for me to get my work done at a very low net cost, largely at the expense of the insurance company.

Sometimes we see a parallel in health insurance. A patient is hospitalized, even though he need not be from the medical point of view, in order to take advantage of hospital insurance which he may have. The hospital profits, the patient likes it and the doctor is readily paid, largely at the expense of the insurance company.

In another situation, a man I know is a salesman traveling on an expense account. His home is rather lower middle class, his family is slightly on the shabby side, his standard of living modest. But when he is on the road he lives like a king. No steak is too high priced for him; no hotel room is above the reach of his employer's pocketbook. Of course, his employer wants him to live well and appear successful, but he takes advantage of this attitude, and maybe the employer does not care too much because it is all deductible in the income tax.

Sometimes we see the same kind of thing in health insurance. Some patients feel that no luxury is too good for them when the insurance company is paying the bill. I remember a case where we were asked to pay for flowers delivered daily to the hospital room.

Another business acquaintance of mine found a way to prolong his stay at a luxurious resort hotel where he had been attending

a convention. It ended Friday, but he stayed over the weekend, in order to make an allegedly worth while business call nearby on a client of his employer's on a Tuesday morning. He admitted to me that the call was of no importance and that he could have gone home Friday night, and that he thought the whole thing a good joke on his company's controller.

A number of times we have had every reason to suspect that a patient stayed in the hospital long beyond the time he needed to do so, because it was comfortable, even luxurious, and we were paying the bill. In other cases we have noticed an apparently undue prolongation of medical treatments. In one case that came to my attention, (and this was one of our own employees) a doctor made two regular visits to a family every week plus some others in between at the office, although the individual insured could give no reason for the calls. The diagnosis shown in the claim forms did not tally with known facts. We were asked to pay $3 for office visits and $5 for home calls, apparently to keep the doctor in luxuries.

As another analogy, I recently had to call on my automobile insurer to reimburse me for extensive repairs required because my wife stopped quickly but the truck behind her did not. I thought the adjuster was checking over the repair bill with extra care, and when I questioned him, he proceeded to unfold a long tale of woe about how repairmen, sometimes with the connivance of, or for the benefit of, clients were padding their bills. It even happened to me — the repairman said, "I will charge for a new bumper, but maybe I can get this one repaired at a $20 'savings' for you after the insurance company pays the bill based on my estimate."

Here we have an analogy that is of exceeding importance. How much bill-padding will there be in health insurance? I have always been one of the first to assert the honesty of the doctors, because I have always felt that they live and work in the highest ethical plane. I find from practical experience, however, that we simply must make sure that the doctors understand what good ethics call for with respect to insurance. I think in many instances it is not a matter of deliberate and conscious violation of ethics on the

part of practitioners, but rather a lack of appreciation of the effect of their attitudes.

We have had numerous examples of the outrageous medical fee, evidently charged because the doctor realized that the insurance company would have to pay all or a large part of it but without full understanding of the long range effects. For instance, in the Equitable we had the case of a truck driver's wife whose surgeon charged $1,500 for a gastrectomy. When we discussed with the doctor the relationship of fees to insurance premiums and the detrimental effect of his charge on the insurance system, the doctor at once proved reasonable and reduced his fee to a more normal level.

This type of outstandingly high and unreasonable fee, while troublesome, is perhaps more easy to deal with and less likely to cause us disaster than a more insidious, general practice, which is less difficult to detect and more difficult to resist — that is the frequent practice of applying relatively small increases or "padding" in fees in a large number of cases where it is recognized that the insurance company either will pay the higher fee, or will pay enough of it so that the patient will not complain. It is in this area of danger of a general "creep-mouse" effect particularly that we feel that we simply must obtain the cooperation of the medical profession generally. Otherwise, comprehensive coverages, such as the highly desirable Major Medical form, will be priced out of the market.

I do not want to give the impression that the insurance companies are niggardly in their attitudes toward health insurance. They do not intend to inflict the doctors with the full weight of the problem of keeping insurance costs low, and they do not intend to browbeat either doctors or patients. Their attitude is rather a positive one. They believe that health insurance is a valuable social mechanism, that it can contribute to the good of all by smoothing out costs over the whole insured population and by improving the credit problem inherent in medicine. On the other hand, insurance must be understood. Everyone concerned must be brought to realize that insurance is not a money-generator. It can do nothing more than re-distribute costs, and it cannot even do that without making a service charge which in effect adds an

expense element to the total medical bill. Therefore, insurance cannot succeed, unless either it adopts rigid safeguards and rather vicious claims practices against costs it did not contemplate covering, or (and this is much the happier way) unless it can achieve cooperation among clients and the medical profession.

Our problem, therefore, is perhaps best characterized as one of communications among men of good will. We must explain insurance. We must recognize its weaknesses as well as its strengths, and we must secure the good will and the cooperation of those that can help us carry on at reasonable costs in spite of the weaknesses. Your cooperation as intermediary between the management of insurance companies and practicing of medicine can be of inestimable value in this respect. I hope you will give sympathetic thought to the opportunities in this direction, and which Mr. Whitehall plans to discuss with you.

MODERATOR HAUGE — Thank you, Mr. Smith.

Our next speaker will be Dr. Percy E. Hopkins, who is a practicing surgeon in Chicago. He also finds time to be very active on various committees of the American Medical Association and his state and county medical societies. He is a Fellow of the American College of Surgeons and a Fellow of the International College of Surgeons. In addition to the Chairmanship of the Prepayment Health Insurance Committee of the American Medical Association, he is also the Chairman of the Committee on Medical Service and Public Relations of the Illinois State Medical Society, former President of the Illinois State Medical Society, and President of the Illinois Medical Service. I am sure that you will find his comments most interesting.

ECONOMICS OF HEALTH CARE

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Committee on Prepayment Medical and Hospital Service of the American Medical Association

It is my understanding that this seminar on medical relations is intended to provide discussions that might be of help in establishing a better relationship between the insurance industry and the members of the medical profession. I specify individual members, rather than medical societies in this connection, because it is necessary to include the individual doctor in any discussion of such relationship. It is the physician in practice who renders the services for which insurance companies have assumed liability, and while it is true that he owes a moral obligation at least to his patient, and is subject to the rules and regulations of the medical society of which he is a member, the physician is entitled and obligated to help form the policies and attitudes of the organizations from the county medical society to the American Medical Association.

What I am trying to emphasize is the fact that while the American Medical Association and the state medical society may develop and recommend general policies regarding insurance as it pertains to the health care field, the whole-hearted participation and the cooperation of the practicing physician is necessary if success is to be achieved. This can be done by means of encouragement, providing of educational information and some patience by both the insurance industry and the medical society. This is necessary if voluntary insurance in this realm is to continue to expand in all directions. I believe the need for growth, both as to numbers insured and benefits provided, is quite urgent if we are to retain independence and our system of free enterprise in this field.

The average doctor in my opinion has no intention of being dishonest, and when he submits a bill greater than his customary fee for services rendered to an insured individual, he probably

just has not stopped to think, or even read, numerous statements in medical journals to the effect that insurance creates no new wealth, nor does the existence of insurance justify an increase in his charges. His attitude may be somewhat influenced by past experiences when many of his bills were questioned in connection with workmen's compensation, or the rendering of service to a victim of an accident. The latter sometimes seemed the occasion for the rendering of bills for larger amounts on the assumption, perhaps, that liability existed in the form of insurance, and he might be able to collect for an occasional one of these cases. Again, he might have been a member of that group of individuals that considers casualty insurance in most instances as something for free; something that does not have to be paid for. Such philosophy, of course, is entirely incorrect and cannot be condoned. It is unfortunate if the physician has to respond to an emergency and never receives compensation, but his creed demands that he render service, and by no manner of interpretation can he justify a position of charging more in another instance than his services are worth. The requirement in times past of frequent filling in of long claim forms, sometimes containing information that was not pertinent to the case, plus the remuneration of his services, such as office calls or dressings, at ridiculously low figures, have been other factors in causing encouragement of overutilization of such services, a feeling of antagonism, and at times even distrust between members of the medical profession and representatives of the insurance industry.

The medical directors of insurance companies for the most part are probably physicians who have been engaged in the insurance field for a considerable period of time, and I believe you will agree that the relationship between members of the medical profession and the insurance industry in the past has left something to be desired. Fortunately, today there is a better understanding of the need for cooperation and willingness on the part of both parties, and much progress has been made in that direction.

Educational efforts are being put forth by the insurance industry and the medical profession in the form of letters, talks, brochures, and meetings to the end that the physician may be informed of his stake in the problem of providing health care on

a voluntary basis. The medical societies are cooperating by wav of inviting criticism and complaints in situations that seem questionable, and have agreed to try to control the actions of their membership, and in this, they are doing the job sincerely, so far as is known.

It may be interesting to note here the effort being made to control the situation in connection with Medicare. While Medicare is in no sense to be considered an insurance program, its operation may be compared to the operation of some of the newer insurance plans. In some states the maximum fees allowable have not been published, and the physician is invited to submit a bill for his usual and customary charges rendered under similar circumstances if the patient were paying the bill. The state societies in such instances have set up committees to consider the bills and are usually able to determine whether or not the charge is a fair one. If it is unrealistic, an effort is made to have the bill reduced, and failing in this, the committee does not recommend payment. Naturally, some protests arise, but it is interesting to note that in one area recently, where a survey was made of several hundred instances in which one particular service was performed, about 70 per cent of the bills rendered were less than the maximum allowable. This seems encouraging, and with some modification might be of help in making larger medical expense plans work more smoothly.

Representatives of the insurance industry have been most cooperative in connection with their simplification of claim forms, and when the use of these forms becomes more general, one of the major complaints of the doctor will have been removed. It may be that sufficient education does not yet exist for the claim forms to have been generally accepted. But it would seem to me that committees from the fields of insurance and medicine can solve the problem to the satisfaction of a large majority of the profession.

In this connection, the question of charges being made by a physician for filling out a claim form should cause no problem. In the event a physician feels that there is justification for a charge, the charge should be made to the person for whom the service is rendered. If a simplified claim form is to be com-

pleted to assist the insured patient to collect a benefit, the physician should consider this as a service to the patient. However, if the physician is asked for a health appraisal for the purpose of underwriting or continuing a risk, then such service should be considered as rendered for the insurance company, rather than the patient.

The fees allowable to physicians for some services rendered in the past were often almost ridiculous because they were so low, and while I would lay no claim to insurance training, and would promptly admit that the benefits in any policy must have a distinct relationship to the premium paid, it is quite easy to see that the insurance company in the minds of the physicians was placed in the unenviable position of expecting to pay less than the customary charge to the patient. The doctor does not stop to reason this out, and there was no one to tell him the facts in the matter.

In a survey of physicians' attitudes toward voluntary health insurance, conducted comparatively recently by the Committee on Prepayment Medical and Hospital Service of the Council on Medical Service of the American Medical Association, it was disclosed that in the opinion of about half of the physicians answering the questionnaire, benefit schedules in many areas were less than the normal charges for low income groups, and about three fourths of the physicians thought the benefits were less than the normal charges for the average income group. However, the benefits seemed closer to the general practitioner's normal charges than those of the specialist. In the same survey, more than half of the physicians who responded expressed the opinion that insured patients are led to assume that their insurance will cover the physician's fee, and more likely the general practitioner's fees, rather than that of a specialist.

Possible remedies suggested were: clearer policies, more thorough reading by the insured, and more thorough explanation by the salesman.

About one fifth of the respondents indicated that their colleagues were likely to charge more than the normal fee for an insured patient, and about one half of these expressed the belief

that the reason was to obtain more money. Others felt that the physician should be compensated for extra trouble or expense involved in handling insured patients.

This survey also disclosed that one fifth of the patients were of the opinion that physicians tend to increase fees for those patients who had insurance coverage. In addition, most physicians believed insurance should cover only a "substantial portion", rather than the entire fee. Over 81 per cent were willing to accept insurance benefits as payment in full for the low income group, but less than 20 per cent were willing to do so for all insured patients, regardless of income.

In the discussion of fees and benefits, a featured special article in current (September) issue of "Monthly Labor Review" is an informative review of the ups and downs in consumer prices for medical care and hospital services between 1936 and 1956. Using the years 1947-49 as a base, the price index for medical care at the close of 1956 was highest of all major items (housing, clothing, and others), just as it is today. But this article points out that if hospitalization is not included in medical care, the price increase for this item between 1936 and 1956 actually is the *smallest of all.*

In this 20 year period, hospital room rates went up 264.8 per cent, which explains why the medical care index has risen so much. At the same time, however, surgeons' fees have gone up only 59.5 per cent, general practitioners' fees 72.8 per cent and dentists' fees 82.1 per cent. This compares with a 220.9 per cent rise for haircuts, 135.0 per cent for shoe repairs and 112.9 per cent for public transportation.

I believe the attitude of the vast majority of physicians in this country has been commendable, but I am aware that there has been a failure to cooperate on the part of some. Unnecessary procedures, prolonged or extended stay in the hospital, increase in charges beyond the normal for services rendered and helping the patient to become eligible for benefits when they would not ordinarily be obtained, constitute some of the more common abuses that have come to my attention. The physician who persists in such practices is either unfamiliar with the purposes and principles of voluntary insurance, or else is very shortsighted.

With the advent of some of the newer types of insurance plans, with more flexibility to meet the legitimate costs of illness, a greater need exists for the honest cooperation of the physician. Failure to cooperate on the part of a large segment of the profession might seriously affect the success of these plans and increase the clamor for the provision of health care by the government.

The medical societies, in general, from the county to the national level, have been aware of the desirability of the voluntary health insurance move, the need for its support and success, as well as the implication of its shortcomings and failure. They also know of the remarkable progress that has been made in recent years in this field, and the continuing effort to provide broader realistic benefits for those in need of health insurance protection. The American Medical Association has stated that it has no intention of giving preferential standing to any one type of plan, but is solicitous that the plans redound to the benefit of the public.

The problem of providing information to a physician is a relatively simple one, but the matter of causing him to become acquainted with that information is another story. The volume of literature sent to physicians by some of the pharmaceutical houses is so great that the matter has even been brought to the attention of the House of Delegates of the American Medical Association. Needless to say, a fair amount of this material is certainly not read. It is known from personal experience that letters from the medical societies, as well as some of the professional journals and periodicals, on occasion also are not read.

Medical society meetings also are not attended as well as might be desirable, except those meetings where a percentage attendance is mandatory. I say this in no critical sense whatsoever, as the average doctor puts in a fairly long day physically and mentally; the scientific subject at a meeting may not be in a field that especially interests him; the meetings in the daytime break up his work, and at night he is frequently too tired to attend a meeting; emergency calls and maternity cases also interfere with attendance; and all in all between department meetings, hospital staff meetings, committee meetings and medical society meetings, he just cannot attend them all and still provide for his family.

However, his hospital staff meetings are a must, as he is required to attend a certain percentage of these meetings. Most of the scientific affairs of the hospital are conducted through departmental meetings, and it is not unusual to occasionally discuss other matters at some of the combined or general hospital staff meetings. It would seem that the hospital staff meeting, or the doctor's office, would be the location where grass-roots contact can best be established with the medical profession. It might take a little time and diplomacy, but I think the contact should and can be made. Medical society meetings should not be excluded, as they too, may be valuable as occasions for the dissemination of information.

The county medical society secretary is probably the key man in connection with the dissemination of information to members of the medical society. It would seem advisable and profitable for all parties that a cordial relationship be established between responsible representatives of the insurance industry organizations and the various county medical society secretaries. If the representatives of the insurance industry happen to be physicians, that too, would be helpful. Incidentally, the question has often arisen in my mind as to whether or not the medical director of an insurance company could spend some time more profitably than to devote at least part of it to medical society organizations, where he could participate as a member and have intimate grass-roots contact with the membership and officers. With a little time and patience, the relationship between the insurance industry and the medical society secretary could be developed into one of mutual trust and cooperation. If contacts between the two parties are limited to those occasions when complaints or grievances are to be aired, the atmosphere may not be so good.

Another field worthy of exploration and serious thought might be to consider the advisability and prospects of the medical directors of insurance companies having representation as an organization in the various county and state medical societies, as well as in the American Medical Association. You might recall that there is a standing committee of the Board of Trustees of the American Medical Association known as the Council on Industrial Health, as well as Sections on Physical Medicine and Preventive

and Industrial Medicine and Public Health. County and state societies have similar committees designed for essentially the same purpose. Many types of insurance must be intimately interlaced in the activities of the people under consideration by this Council and these committees.

Representatives of the Health Insurance Council have appeared at some of the meetings of the Committee on Prepayment Medical and Hospital Service, and it is hoped this practice will continue. Pertinent problems are discussed, and it is intended that these meetings will continue to be productive and beneficial. Some articles by men engaged in the health insurance industry have been submitted to the Editor of the *Journal of the American Medical Association* with the recommendation that they be published because of their educational value. The medical profession must depend upon men trained in insurance for the development of legitimate and realistic health insurance plans that will keep pace with the needs of the public. The medical profession can be depended upon to provide the professional services needed, promptly, efficiently and honestly.

To accomplish this, a continuing program of education of the medical profession and the public is necessary. Considerable progress has been made, but it must be a continuing effort expanded wherever and however possible. Grievance committees, or the equivalent, should be utilized to bring to the attention of the medical society when necessary, flagrant violations or abuses that occur, with the acknowledged responsibility of the society to discipline the member. The individual physician must be told how great is his stake in the project of voluntary health insurance and of some of the things that might occur if he is no longer able to practice as a free individual. The public should be informed of the principles of insurance and as to the benefits provided in their contracts. Some way should be found to explain the consequences of the abuse of their contracts. The inconsistency of some labor officials in their demands for unrealistic benefits and principles that do not coincide with our democratic way of life should be pointed out.

Finally, realizing that we are living under a system of free enterprise, under which system we have become the greatest nation

in the world, we as physicians and men dedicated to a cause, have at heart the interests and welfare of the public. If this premise is true, we could well cooperate in the consideration of proposed legislation that affects the provision of health care to the public. The history of the medical profession in this country is one of honor, and one that has never been dependent upon, nor a ward of, the government. It has been a supporter of, and a contributor to, the government. It has responded in every crisis. The health record of this country is better than that of any large country, and probably most small countries. The medical profession is firm in its intention and desire to remain free in caring for the sick. The plan of voluntary insurance affords the medical profession and the insurance industry another opportunity to demonstrate to the nation that we have no need for a government-owned and regulated project that has no place in this country.

MODERATOR HAUGE — Thank you very much, Dr. Hopkins. Your comments, and particularly your sound, constructive recommendations, are most thought-provoking.

Our final speaker this morning is Mr. Albert V. Whitehall. Mr. Whitehall is an attorney who practiced law in Chicago for eleven years. For eight years of his legal practice, he was a staff attorney for the *Chicago Daily News*, and then, for nine years, represented the American Hospital Association in Washington, D. C., as Director of its Washington Service Bureau. For two years, he was Executive Secretary of the Blue Cross Plan in Seattle, Washington. In 1956, he became Associate Director of Health Insurance of the Life Insurance Association of America, and, this spring, he was elected Vice Chairman of the Health Insurance Council.

THE VOLUNTARY HEALTH INSURANCE PARTNERSHIP

ALBERT V. WHITEHALL
Vice Chairman, Health Insurance Council,
and
Director of Health Insurance,
Life Insurance Association of America

The Impact of Health Insurance

Within our own generation, we have been learning to use the stabilizing force of the insurance mechanism in a new field — health care. The revolution in medical science has brought health care and insurance together. Life insurance, which is owned by 86 per cent of all American families, has largely conquered dependency of widows and orphans. Casualty insurance has made it possible for small business and individuals to endure the normal hazards of our free enterprise system and still survive. But no use of the insurance mechanism has called for more ingenuity, flexibility and imagination than voluntary health insurance.

Today, no family would willingly return to that era when a costly illness could plunge a family into poverty. No doctor really wants to go back to the days when his bills often were paid in pigs and potatoes or other barter — or perhaps not paid at all. Health insurance is here to stay in spite of the problems inherent in adjusting the rigid insurance mechanism to the health field, where the only constant factor is change. It has demonstrated the contribution it can make to human welfare.

Health insurance is already big business. In 1956 it paid out through:

Life insurance companies	$1.7 billion
Casualty and other insurance companies	.4 "
Independent programs	.1 "
Blue Cross-Blue Shield	1.4 "
A total of	$3.6 billion

Compare this to life insurance payments of $2.4 billion in death benefits, and $3.5 billion in living benefits such as annuities, dividends, matured endowments, and disability benefits, and you will

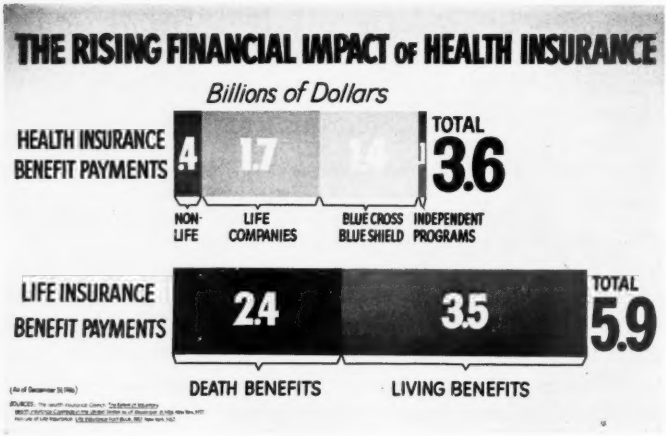

CHART 1

see that health insurance handles a lot of money. These figures are growing, too, as broader benefits are brought to more people.

Health insurance is big business socially. At the end of 1956, 116 million of our 170 million population had some form of health insurance protection. This is nearly 70 per cent of the population. Excluding unknown numbers not even eligible for health insurance, such as men in military hospitals, indigent veterans, inmates of homes for the aged, mental institutions, and many others, the proportion is even larger. Compare the 116 million with health insurance against the 70 million wage earners under social security, and the 118 million persons who own some form of life insurance and you see that the influence of health insurance upon our way of life is very great.

Health insurance is big business politically. It is almost inevitable that any social phenomenon that affects large numbers of people, sooner or later comes under political scrutiny — and government regulation. Everyone is aware that health insurance is under political scrutiny here and abroad. It has become a government monopoly abroad; in this country voluntary health insurance has been the

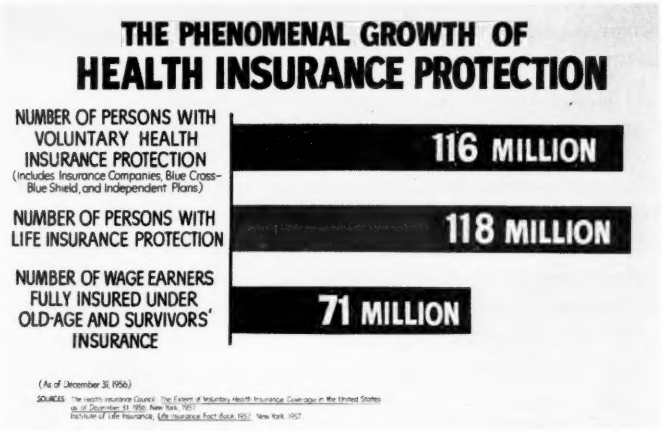

CHART 2

strongest single factor in holding off the government monopoly called socialized medicine.

Voluntary health insurance will continue to serve the American people just so long as the people are convinced that we serve them better than any other way. This is a real challenge to those of us who believe in our "voluntary — free enterprise" way of life.

The Partnership

The very name "Health Insurance" suggests that in its service to the American people, it is a partnership between two very old and highly respected segments of our economy: the health care field and the insurance business. As in any partnership, each partner depends upon the other for mutual cooperation.

Without the voluntary health insurance mechanism, doctors might be working for a single, monopolistic employer, probably the federal government. They might even be discussing how to get a wage increase, as their British brethren recently did.

On the other hand, the insurance mechanism is quite literally at

the mercy of its partner — the health care field, and particularly the medical profession. It is not unfair to say that health is the senior partner in health insurance — it is certainly the controlling partner.

The insurance company simply collects health care dollars into a common pool and pays it out according to very rigid rules. But, who is the partner with his hand on the spigot? Who regulates the flow of health insurance dollars? It is the doctor. Every claim requires a certification of a doctor's professional judgment.

The doctor's professional judgment is often exercised under considerable pressure. At best, it involves subjective factors and influences.

> In a fire insurance claim, the loss can be measured.
>
> In a life insurance claim, a man is either dead or he is not.
>
> But in a health insurance claim, how sick *is* the patient?
>
> Is an eloquent hypochondriac entitled to more benefits than a stoic?

For examples of the pressures that doctors face in making these decisions, consider these:

> The patient who wants to stay an extra day in the hospital "because I have insurance, doctor, and it will not really cost any more."
>
> The patient who says, "Doctor, if you will put me in the hospital my insurance will pay your fee for that toenail operation."
>
> The patient who says, "Doctor, I have major medical insurance, and if you will make your bill 25 per cent higher, I will get enough to pay you in full and you will not have to wait."
>
> The doctor who says to himself, "That patient has insurance so he is well able to pay a little extra fee; and besides, my services are worth it."
>
> The doctor who believes the insurance business has some sort of pipeline from Fort Knox; and that if *he* had all the money the insurance companies have, *he* would not be so petty about a minor increase in fees.
>
> Or the doctor who has spent hours filling out 157

varieties of health insurance forms — all of them simple, but all of them different! What frame of mind is *he* in?

These are *only a few* of the pressures upon the practicing physician.

We need to know about them; we need to understand them. We need to support our partners — the doctors — in standing up under these pressures so they can make the sort of objective, professional judgments we must have, if health insurance is to remain voluntary.

The daily decisions of the practicing physician influence the total cost of health care. They also influence the attitude of the general public toward the present freedom from governmental regulation which medicine enjoys and which we want to preserve.

So now our problem is how to make the practicing physician aware of the cumulative consequences of his decisions as they influence the total cost of health care. He is entitled to know the financial, the sociological, the political influences of his decisions. He should know, for example, that:

> Insurance is merely a stabilizer; it does not create more money. Insurance collects money and administers it, but the *doctor's professional judgment* carries the responsibility for its expenditure. There is real danger of inflating the cost of voluntary health insurance beyond public tolerance.

The Health Insurance Council

More than ten years ago, insurance leaders recognized that the "partners in health insurance" *must* talk to each other and learn to work together in joint service to the public. They set up the Health Insurance Council for that purpose.

To avoid improper emphasis upon any single segment of the insurance business, the Council was made a federation of several existing associations. The insurance business is highly competitive, and because competition produces progress, we want to keep it that way. However, there should be no competition within the Council; our interests are identical and broad. We should speak with one voice for the insurance business as a whole on these vital

issues. The federation concept should lift us above our competitive pressures as we try to tell the health insurance story to the producers of health services who are our partners in service to the public.

This is not the place to discuss the machinery of the Council, nor how it draws its committees from all segments of the industry. It is proper to say, however, that the Council has become an effective voice for the industry as a whole, in communications with leaders of the hospital field and the medical profession.

The Board of Trustees of the American Hospital Association more than two years ago gave formal approval to our hospital admission forms. There have been many indications of improved understanding with hospital leaders, as they learn of our improved service to them.

Members of the Board of Trustees of the American Medical Association met this summer with presidents and medical directors of several insurance companies to discuss how we could make voluntary health insurance serve the public better. It was agreed there that one of our principal problems is to bring a better understanding of health insurance principles to the medical profession at the practicing physician level. We promised we would do it. I am proud to say that our program has already been launched.

The Communications Program

Our nation has nearly 200,000 doctors. We will never reach them all if we depend entirely on the staff of our member associations. But doctors are in daily contact right now with insurance. For example, workmen's compensation, health insurance claim forms, life insurance examinations are only a few of the contacts.

Both the insurance business and the medical profession contribute their share of good citizens to civic and community life. These men meet each other in church work, community chest drives, hospital trusteeships, politics — even fishing and the golf course.

Practicing physicians as a class are well educated, high earning, family men who believe in the ownership of life insurance to protect their families and to build their estates. Does it seem possible

that our carefully selected, intelligently trained life insurance agents have not sought them out?

Last, but not least, every life insurance company requires administrative advice through full or part time medical directors. You gentlemen have a unique bond with your colleagues in practice. You belong to your local medical society, you maintain your interest in clinical medicine, you are interested in maintaining the free enterprise system in medicine and the insurance business. You can help. In fact, it has been repeatedly brought home to me that the life insurance company medical director is one of our best, most effective channels of communication between the insurance business and the medical profession. You *must* help. You are essential.

Now, our program has been launched. Here is what it is and how you can fit into it.

In each state we are designating a state chairman to organize Health Insurance Council activities. We have asked all the companies to suggest men of stature and competence to volunteer in helping us carry the health insurance story to doctors and hospitals. The reaction of companies has been splendid. Many have told us "We have been waiting for you to ask us to help."

I suspect that many of you feel that way, too. The participation of your Association in the activities of the Health Insurance Council over the years has been one of our sources of strength. Here is a program where you can each participate, within the limits of your own busy lives, but with great personal satisfaction and effectiveness. We ask that you seek out your state chairman and volunteer your help and support. If you do not know who he is, ask us.

We are urging that state and county medical societies set up committees to meet with our local committees. We urge, too, that physicians be invited to address insurance gatherings and that insurance men appear on programs of state and county medical society meetings, and hospital staff meetings. We are offering articles by competent authorities in the insurance field for publication in both medical and insurance journals, and we invite short, simple articles on health insurance as it affects the doctor from any of you who would like to submit them.

Of course, we expect the Health Insurance Council staff to help the programs get organized and to assist and guide their development. We hope to provide helpful material — speakers' kits and aids. If you are a member of a state team, we will put you on our mailing list to receive this material. And we shall want reports from you of what you do.

We do not suggest any single, rigid pattern of state committee activity. Your state committee must meet the needs it finds. We do offer some examples and some precautions:

> In Wisconsin, two years ago, a local company home office man made a tour of a dozen or more county societies. He got the support of the state society secretary who made the tour with him. We have that man's excellent notes of his experience.
>
> In Pennsylvania, we are getting a speaker's program under way, with several invitations already in from county societies.
>
> In California, we have a team in the field. It includes both lay people and doctors, and they have already made a fine beginning on an educational program, particularly with articles in county society bulletins.
>
> In New Jersey, we have a committee of insurance people that has already developed a fine, friendly liaison with the medical society and the hospitals.
>
> In Texas, the famous HIPJAC Committee was set up some years ago and still functions. We hope to enlist that Committee in our program.

In some states, we have negotiated service-type programs with state medical societies. Here is my first caution: We earnestly urge that you avoid becoming involved in discussions of details and technicalities of specific programs. The negotiation of benefits, rates, interpretations of contracts, and so forth, require specially authorized committees for that purpose. But you can open doors. You can tell our story, being sure you know it first, and you can listen.

And here is my second caution: Do not expect miracles to happen overnight. Insurance and medicine are going to live together a long time. Place the emphasis on making it a lasting friendship

— an effective partnership that will serve the American public well.

The Strength of Voluntary Health Insurance

In conclusion, one brief reference to a basic economic principle that is important to the insurance business, to the medical profession and to our free enterprise system — competition. The insurance business is highly competitive; a friend of mine has described the effort of the Health Insurance Council to develop a uniform program for the 800 companies writing health insurance as trying to put 800 cats in a bag.

Our 800 insurance companies compete vigorously for the right to serve the American people. Under our system, they do it by offering something better. We believe that if the American people are free to choose, and adequately informed, they will choose the best. When something better comes along they will choose that.

The practicing physician believes in freedom of choice, too. The sacred doctor-patient relationship includes the knowledge that if the doctor does not do his very best, the patient is free to choose another doctor.

It is this pressure for progress that makes our free enterprise system strong. It is our people who benefit from it. And it is we who must always remember that voluntary health insurance can only survive as long as it serves.

We in the insurance business, and our partners in the health care field, must work together to keep health care and health insurance voluntary so that these pressures for progress may continue.

MODERATOR HAUGE — Thank you, Mr. Whitehall.

If this discussion will stimulate the members of our organization to become enthusiastic supporters of the program outlined by Albert Whitehall, I feel that our time will have been well spent.

Now, we had intended to provide a short period for questions but our time is very short. Dr. Getman, you may take over.

PRESIDENT GETMAN — Thank you, Dr. Hauge, Mr. Smith, Dr. Hopkins, and Mr. Whitehall. I am sure that we all appreciated your remarks. You gave us a great deal of material to think over.

At this time, I think, we can set aside just a half minute, if you will, so that I may express my thanks to all those who have helped me during this meeting, and in the year preceding it. I would like, first, to thank the Program Committee, of which Dr. Bell was Chairman; Dr. John Peck and Dr. Charles Kiessling; our Convention Committee, of which Dr. John J. Hutchinson was Chairman; and so ably assisted by Dr. David M. Benford and Dr. Royal S. Schaaf.

In addition, I should also like to thank Mrs. William Bolt, Chairman of the Ladies' Committee, and her assistants, Mrs. Henry Kirkland and Mrs. Norman G. Barker, and also those other ladies who so ably assisted them, Mrs. Richard Willis, Mrs. David Benford, and Mrs. John Hutchinson. As you know, this committee was something of an innovation in our New York meeting, and I hope that your wives have enjoyed the opportunity afforded by this group.

I would be very remiss indeed if I did not thank Dr. Schaaf, in his new position as Secretary, for his loyal help through the past year. And, last, our good friend, Miss Clara Rizzolo, who so ably takes care of all of us every year. She was assisted this year, I think, most ably, and I want to thank them for their time, by Miss Florence Dressler and Miss Helen Schaeffer.

Lastly, I should like to thank you for allowing me to preside at this meeting. It has been a great pleasure, indeed.

And now, I would like to introduce to you your new officers, first, those reelected:

Dr. James Gudger, Editor of the Transactions.

Dr. J. Grant Irving, Treasurer.

Your new Vice President, Dr. Ennion S. Williams.

Your President-Elect, Dr. Henry B. Kirkland.

And now, with the greatest pleasure, I turn this gavel over to my friend, Dr. Norman J. Barker.

(President Barker assumed the Chair.)

PRESIDENT BARKER — Members of the Association and Guests: Thank you for this wonderful expression of your confidence in me. I am profoundly moved by the honor and privilege of being chosen to serve as your President, as well as the responsibility

involved. I am looking forward to the coming year, to the opportunity it will afford me to work with you toward the solution of some of our mutual problems. I think that our most immediate challenge will be to try to match the splendid program that has just been presented by your retiring President, Dr. Getman, and his associates.

Subject to the approval of the Executive Council, I would like to hold the next meeting of this Association in Hartford. The tentative dates are October 22, 23 and 24, 1958. We have a fine new Statler Hotel in Hartford for your accommodation and convenience, and Connecticut General has a beautiful new home office building that we want you all to visit. The weather is usually fine at this time of the year, and New England can be very lovely. I hope to see you all in Hartford next fall, and I hope that you will be accompanied by your wives, for whom we are going to plan a very special program.

Mr. Secretary, is there any other business to come before the meeting?

DR. YLVISAKER — Mr. President, I am sure we are all agreed that we have had a wonderful meeting and that we are all indebted to Dr. Getman for the splendid leadership he has given the Association for the last year, and for the wonderful arrangements he made for this meeting, as well as the pleasant way in which he has conducted the sessions.

I would also like to join with him in thanking all the other committees which have helped him in making the meeting as successful as it has been.

I am sure that we would all want to rise and join in a round of applause in tribute for the wonderful work that he has done.

PRESIDENT BARKER — I am sure that we all concur in the sentiments expressed by Dr. Ylvisaker, and the Secretary will include this resolution in the Transactions.

Is there any other business to come before us? If there is no further business to come before the meeting, a motion to adjourn is in order.

Upon motion regularly made and seconded, it was unanimously voted to adjourn the meeting.

The following doctors were present at some time during the sessions:

F. B. Agee, Jr.
C. B. Ahlefeld
A. B. Ainley
A. C. Albright
V. G. Allport
Henry Almond
Joseph Altman
N. L. Armstrong
A. B. Ayers

C. P. Bailey
N. J. Barker
G. P. Barnett
C. M. Barrett
S. F. Bassett
C. C. Beach
E. F. Beach
J. R. Beard
M. F. Bell
M. B. Bender
D. M. Benford
W. R. Bishop
H. W. Blackburn
J. E. Boland
William Bolt
E. C. Bonnett
D. T. Book
M. T. Boss
J. R. Bowen
H. J. Brekke
E. J. Brogan
A. W. Bromer
A. E. Brown
H. B. Brown
R. F. Brown
R. F. Buchan
J. B. Bullock

D. G. Cameron
D. B. Campbell
R. L. Candage
A. W. Capon
N. S. Clark
W. A. Clarke
R. B. Cleveland
N. B. Cole
G. R. Collyer
H. L. Colombo
B. R. Comeau
F. R. Congdon
J. L. Cook
R. H. Craig

J. P. Davis
B. A. Dawber
F. E. Demarais
F. R. Dieuaide
A. H. Domm
R. M. Donauer
J. P. Donelan
G. D. Dorman
L. B. Dunn

W. W. Eakin
L. H. Earle, Jr.
T. M. Ebers
L. B. Ellis
L. J. Emanuele
P. S. Entmacher

A. H. Faber
R. B. Failey, Jr.
J. G. Falconer
R. K. Farnham
R. W. Finegan
M. W. Fischbach
L. F. Flick
J. G. Forgerson
P. M. L. Forsberg
E. M. Freeland
C. E. Fronk

J. O. Gagnon
F. I. Ganot
J. T. Geiger
D. B. Gelfond
J. P. Gemmell
E. E. Getman
F. W. Gluck
R. A. Goodell
George Goodkin
H. M. Goodman
H. W. Goos
J. K. Gordon
R. S. Gordon
C. D. Gossage
A. L. Grasmick
Ghent Graves
A. C. Grunow
J. R. Gudger
V. W. Gunter

Llewellyn Hall
G. W. Halpenny
V. G. Hammond
O. E. Hanes
J. A. A. Harcourt
O. C. Hardwig
J. R. Harnes
F. F. Harris
L. E. Hathaway, Jr.
H. L. Hauge
W. C. Hausheer
M. H. Henderson
O. C. Hendrix
R. E. Henning
H. H. Hershey
E. V. Higgins
Arthur Hill
W. H. Hill
E. C. Hillman, Jr.
P. E. Hopkins
J. C. Horan
E. G. Howe
T. B. Hoxie
A. A. Humphreys
J. L. Humphreys
J. H. Humphries
B. L. Huntington
J. R. B. Hutchinson
J. J. Hutchinson

A. S. Irving
J. G. Irving

D. S. James
A. N. Jay
F. E. Jenkins
T. J. Jernigan
J. W. Johnson, Jr.
R. M. Johnson
Nathaniel Jones

V. L. Karren
E. A. Keenleyside
N. R. Kelley
E. F. Kerby
N. C. Kiefer
C. E. Kiessling
Richard King
C. T. Kirchmaier
H. B. Kirkland
C. E. Kossmann
Edward Kuck
Paul Kurzweg, Jr.

Phillips Lambkin
P. H. Langner, Jr.
W. F. Larrabee, Jr.
A. L. Larson
Timothy Lee
H. R. Leffingwell
R. J. Lempke
W. R. Leute, Jr.
J. C. Lindner
E. H. Lindstrom
R. P. Lockhead
J. F. Lovejoy
W. C. Lowery

F. M. McChesney
M. G. MacDonald
A. J. McGanity

J. D. McGaughey, III
F. J. McGurl
T. J. McGurl
C. D. McKeown
George McLean
L. L. McLellan
W. M. McMillan
W. J. McNamara
Morton Magiday
S. J. Newton Magwood
John Malgieri
P. V. Martin
F. A. L. Mathewson
F. D. Mauk
P. S. Metzger
L. C. Miller
M. B. Miller
I. G. Milne
R. M. Miskimon
G. S. Modjeska
R. C. Montgomery
J. F. Moore, Jr.
S. R. Moore
A. W. Morrow
C. V. Mulligan
J. R. Murphy
Sidney Narins
R. M. Nay
M. H. Neill
R. E. Nicholson
R. D. O'Connor
R. J. Oehrig
W. F. H. O'Neill
E. T. Opstad
J. K. T. Ormrod
A. E. Parks
J. McC. Peck
D. S. Pepper
J. I. Peters, Jr.
R. W. Peterson
J. C. Pierson
F. I. Pitkin
T. E. Plucinski
A. A. Pollack
H. E. Pugsley
W. O. Purdy
R. S. A. Purkis
M. A. Puzak
J. H. Ready
C. L. Reeder
P. V. Reinartz
W. M. Reynolds
Paul Reznikoff
G. P. Robb
D. C. Roberts
A. J. Robinson
J. C. Robinson
H. B. Rollins
R. C. Roskelley
E. S. Ross
M. E. Rougraff
B. A. Ruggieri
N. R. Ruud
M. T. Ryman
D. Y. Sage
R. S. Schaaf
K. F. Schaefer
L. P. Schroeder
P. C. Schwager
B. T. D. Schwarz
W. H. Scoins
R. C. Secor
A. F. Seibert
D. L. Selby
T. S. Sexton
Hall Shannon
Daniel Sheehan
E. B. Shepherd
J. T. Sheridan
V. T. Shipley
R. R. Simmons
V. P. Simmons
W. H. Simmons
G. A. Simpson
H. N. Simpson
J. R. Slamer
N. H. Sloan
D. E. Small
W. A. Smith
F. L. Springer
H. F. Starr
H. F. Starr, Jr.
D. F. R. Steuart
L. Q. Stewart
F. M. Stites
Robert Stock
Lee Stover
J. C. Talbot
M. J. Taylor
L. J. Tedesco
G. F. Tegtmeyer
L. S. Thompson
W. C. Thompson
W. B. Thornton
Joseph Travenick, Jr.
Wallace Troup
F. D. Truax
Maurice Turcotte
H. E. Ungerleider
R. C. Voss
P. C. Waldo
Peter Walsh
K. E. Ward
R. V. Ward
F. A. Warner
C. F. Warren
G. H. Watters
R. L. Weaver
Jefferson Weed
J. L. Weinstock
S. S. Werth
G. H. White
C. W. Whitmore
J. A. Wilhelm
R. W. Wilkins
E. S. Williams
R. L. Willis
A. C. Wilson
S. G. Wilson
D. H. Woodhouse
L. S. Ylvisaker
D. E. Yochem
G. G. Young
R. W. Zinkann

Also present were:

J. F. Anderson
T. L. Blalock
C. C. Bryan
E. G. Bullis
R. A. Burke
J. B. Corbett
Owen Cullimore
T. H. Leath
Herbert Marks
H. A. Moreen
P. G. Ochterbeck
L. N. Parker
B. S. Pauley
Morris Pitler
H. C. Reed
Harris Scherman
Pearce Shepherd
O. G. Sherman
J. H. Smith
W. M. Stufflebeem
R. J. Vanderbeck
A. C. Webster
A. V. Whitehall
P. C. Wickens
J. C. Wilberding
Miss A. M. Lyle
Miss Hazel Wallace

Total attendance at all sessions, 322.

In Memoriam

Deceased since the Sixty-fifth Annual Meeting

Edgar W. Beckwith, M. D.
The Equitable Life Assurance Society of the United States, New York City
Died September 24, 1957

Harry W. Dingman, M. D.
Continental Assurance Company, Chicago, Ill.
Died September 30, 1957

William M. Genthner, M. D.
Continental American Life Insurance Company, Wilmington, Del.
Died April 4, 1957

Byam Hollings, M. D.
John Hancock Mutual Life Insurance Company, Boston, Mass.
Died January 25, 1957

Herbert B. Kennedy, M. D.
Woodmen of the World Life Insurance Society, Omaha, Neb.
Died August 22, 1957

Ralph E. McLochlin, M. D.
National Old Line Insurance Company, Little Rock, Ark.
Died November 8, 1957

Herbert Old, M. D.
Provident Mutual Life Insurance Company, Philadelphia, Pa.
Died May 24, 1957

Hector M. Stevenson, M. D.
Aetna Life Insurance Company, Hartford, Conn.

LIST OF MEMBERS OF THE ASSOCIATION OF LIFE INSURANCE MEDICAL DIRECTORS OF AMERICA

Fred B. Agee, Jr., M. D.	Aetna, Hartford, Conn.
Charles B. Ahlefeld, M. D.	Business Men's, Kansas City, Mo.
Allan B. Ainley, M. D.	Travelers, Hartford, Conn.
Arthur C. Albright, M. D.	Victory Mutual, Chicago, Ill.
Henry Almond, M. D.	Metropolitan, New York City
Joseph Altman, M. D.	Companion Life, New York City
Donald A. Anderson, M. D.	Washington National, Evanston, Ill.
Frank R. Anderson, M. D.	Pacific Mutual, Los Angeles, Calif.
Karl W. Anderson, M. D.	Northwestern National, Minneapolis, Minn.
Perry A. Anderson, M. D.	Rockford Life, Rockford, Ill.
Nolen L. Armstrong, M. D.	United Founders, Oklahoma City, Okla.
Thomas M. Armstrong, M. D.	Philadelphia Life, Philadelphia, Pa.
Bernard Baillargeon, M. D.	Alliance Nationale, Montreal, Canada
John W. Barch, M. D.	Lincoln National, Fort Wayne, Ind.
William S. Barcus, M. D.	Pioneer American, Ft. Worth, Tex.
Norman J. Barker, M. D.	Connecticut General, Hartford, Conn.
Gordon P. Barnett, M. D.	Kansas City Life, Kansas City, Mo.
Charles M. Barrett, M. D.	Western and Southern, Cincinnati, Ohio

G. Robert Bartron, M. D.	Midland National, Watertown, S. D.
Samuel F. Bassett, M. D.	Prudential, Newark, N. J.
Robert W. Bates, M. D.	Manufacturers, Toronto, Canada
Eliot F. Beach, Ph. D.	Metropolitan, New York City
Irwin W. Bean, M. D.	Fidelity, Regina, Can.
J. Randolph Beard, M. D.	Mutual Benefit, Newark, N. J.
James E. Bee, M. D.	Kansas City Life, Kansas City, Mo.
Murray F. Bell, M. D.	New York Life, New York City
Maurice B. Bender, M. D.	Guardian, New York City
David M. Benford, M. D.	Metropolitan, New York City
Robert A. Benson, M. D.	Metropolitan, Ottawa, Canada
Roy W. Benton, M. D.	Northwestern Mutual, Milwaukee, Wis.
C. Coleman Berwick, M. D.	Metropolitan, San Francisco, Calif.
Francis P. Bicknell, M. D.	State Mutual, Worcester, Mass.
B. Cosby Bird, M. D.	Preferred, Montgomery, Ala.
William R. Bishop, M. D.	Provident Life Acc., Chattanooga, Tenn.
John E. Boland, M. D.	Country, Chicago, Ill.
William Bolt, M. D.	New York Life, New York City
John M. Bond, M. D.	Northwestern Mutual, Milwaukee, Wis.
Earl C. Bonnett, M. D.	Metropolitan, New York City
M. Theodore Boss, M. D.	Home Mutual, Baltimore, Md.
John R. Bowen, M. D.	Penn Mutual, Philadelphia, Pa.
J. Thornley Bowman, M. D.	London Life, London, Canada
Ernest L. Boylen, M. D.	Standard, Portland, Ore.

William R. Bradley, M. D.	Phoenix Mutual, Hartford, Conn.
Kenneth F. Brandon, M. D.	Aetna, Hartford, Conn.
Warren C. Breidenbach, M. D.	California Life, Oakland, Calif.
David J. Breithaupt, M. D.	Manufacturers, Toronto, Canada
Harvey J. Brekke, M. D.	Lutheran Brotherhood, Minneapolis, Minn.
Robert D. Brewer, Jr., M. D.	Prudential, Chicago, Ill.
Harry Brodsky, M. D.	Beneficial Standard, Los Angeles, Calif.
Edmund J. Brogan, M. D.	Provident Mutual, Philadelphia, Pa.
Albert W. Bromer, M. D.	Metropolitan, New York City
Arthur E. Brown, M. D.	New England Mutual, Boston, Mass.
C. Alton Brown, M. D.	Home State, Oklahoma City, Okla.
C. Frank Brown, M. D.	Southwestern, Dallas, Tex.
Howard B. Brown, M. D.	Massachusetts Mutual, Springfield, Mass.
Ronald F. Buchan, M. D.	Prudential, Newark, N. J.
John B. Bullock, M. D.	Life Insurance Co. of Virginia, Richmond, Va.
Carroll A. Burroughs, M. D.	Peoples, Frankfort, Ind.
W. Lawrence Cahall, Jr., M. D.	John Hancock Mutual, Boston, Mass.
Douglas G. Cameron, M. D.	Prudential Assurance, Montreal, Can.
Douglas B. Campbell, M. D.	National, Toronto, Can.
Raymond L. Candage, M. D.	State Mutual, Worcester, Mass.
David W. Carter, Jr., M. D.	Reserve Life, Dallas, Tex.
Paul H. Charlton, M. D.	Midland Mutual, Columbus, Ohio

Harry E. Christensen, M. D.	Union Mutual, Portland, Me.
Norman S. Clark, M. D.	Independent Order of Foresters, Toronto, Can.
William A. Clarke, M. D.	Travelers, Hartford, Conn.
Howard R. Clement, M. D.	John Hancock Mutual, Boston, Mass.
Robert B. Cleveland, M. D.	Equitable Life Assurance, New York City
Milton H. Clifford, M. D.	New England Mutual, Boston, Mass.
Harry A. Cochran, Jr., M. D.	Lincoln National, Fort Wayne, Ind.
John E. C. Cole, M. D.	North American, Toronto, Canada
Norman B. Cole, M. D.	Baltimore Life, Baltimore, Md.
Irwin E. Colgin, M. D.	Texas Life, Waco, Tex.
G. R. Collyer, M. D.	London Life, London, Canada
Harry L. Colombo, M. D.	National Life, Montpelier, Vt.
Berthold R. Comeau, M. D.	Northeastern, New York City
Frederick R. Congdon, M. D.	Berkshire, Pittsfield, Mass.
Chester E. Cook, M. D.	Southwestern, Dallas, Tex.
J. Lindsay Cook, M. D.	Pilot, Greensboro, N. C.
Robert H. Craig, M. D.	Prudential, Newark, N. J.
Neil L. Criss, M. D.	United Benefit, Omaha, Neb.
Henry A. Cromwell, M. D.	American Capitol, Houston, Tex.
Howard K. Crutcher, M. D.	United Fidelity, Dallas, Tex.
Khurshed J. J. Cursetji, M. D.	Life Insurance Corporation, Bombay, India
John P. Davis, M. D.	Security Life & Trust, Winston-Salem, N. C.

Bryan A. Dawber, M. D.	Penn Mutual, Philadelphia, Pa.
John S. Delahaye, M. D.	Empire Life, Kingston, Canada
Harold D. Delamere, M. D.	Crown, Toronto, Canada
Aniceto Del Rio, M. D.	La Nacional, Mexico City, Mexico
Earle T. Dewey, M. D.	Metropolitan, San Francisco, Calif.
John A. Dillon, M. D.	Puritan, Providence, R. I.
Fred Dinkler, M. D.	Great Southern, Houston, Tex.
Albert H. Domm, M. D.	Prudential, Los Angeles, Calif.
Robert M. Donauer, M. D.	Prudential, Newark, N. J.
James P. Donelan, M. D.	Guarantee Mutual, Omaha, Neb.
Verner J. Donnelly, M. D.	Prudential, Houston, Tex.
Gerald D. Dorman, M. D.	New York Life, New York City
James T. Downs, Jr., M. D.	Fidelity Union, Dallas, Tex.
Raymond L. Dross, M. D.	Prudential, Houston, Tex.
Thomas C. Dunlop, M. D.	Manufacturers, Toronto, Canada
Louis B. Dunn, M. D.	Postal, New York City
William W. Eakin, M. D.	Standard, Montreal, Canada
Lyon H. Earle, Jr., M. D.	Connecticut General, Hartford, Conn.
Theodore M. Ebers, M. D.	Connecticut Mutual, Hartford, Conn.
Rolf K. Eggers, M. D.	General Life, Seattle, Wash.
Laurence B. Ellis, M. D.	Boston Mutual, Boston, Mass.
Louis J. Emanuele, M. D.	New York Life, New York City
James C. Emmett, M. D.	Imperial, Toronto, Canada

Jack A. End, M. D.	Northwestern Mutual, Milwaukee, Wis.
Paul S. Entmacher, M. D.	Metropolitan, New York City
Albert H. Faber, M. D.	New York Life, New York City
J. Gilbert Falconer, M. D.	North American, Toronto, Canada
Raymond K. Farnham, M. D.	Metropolitan, New York City
William S. Fewell, M. D.	Liberty, Greenville, S. C.
Ralph M. Filson, M. D.	Travelers, Hartford, Conn.
Rexford W. Finegan, M. D.	Metropolitan, New York City
Frederick Fink, M. D.	National Bankers, Dallas, Texas
Max W. Fischbach, M. D.	Plymouth Mutual, Philadelphia, Pa.
Lawrence F. Flick, M. D.	Provident Indemnity, Norristown, Pa.
James G. Forgerson, M. D.	Metropolitan, San Francisco, Calif.
Philip M. L. Forsberg, M. D.	United Life and Accident, Concord, N. H.
Garth E. Fort, M. D.	National Life & Accident, Nashville, Tenn.
Arthur C. Fortney, M. D.	Western States, Fargo, N. D.
John M. Foster, M. D.	Capitol, Denver, Colo.
John T. France, M. D.	State Farm, Bloomington, Ill.
Edward M. Freeland, M. D.	New York Life, New York City
Clarence E. Fronk, M. D.	Hawaiian Life, Honolulu, T. H.
Robert E. Funke, M. D.	Prudential, Los Angeles, Calif.
F. Irving Ganot, M. D.	Prudential, Newark, N. J.
David S. Garner, M. D.	Shenandoah, Roanoke, Va.

Duke R. Gaskins, M. D.	Hospital Benefit Assur., Phoenix, Ariz.
J. H. Geddes, M. D.	Northern, London, Canada
John T. Geiger, M. D.	Metropolitan, New York City
D. Barton Gelfond, M. D.	Philadelphia Life, Philadelphia, Pa.
John P. Gemmell, M. D.	Monarch, Winnipeg, Canada
Edson E. Getman, M. D.	New York Life, New York City
Charles A. Gianasi, M. D.	Continental Assurance, Chicago, Ill.
Edgar G. Givhan, Jr., M. D.	Protective, Birmingham, Ala.
Francis W. Gluck, M. D.	Monumental, Baltimore, Md.
Robert A. Goodell, M. D.	Phoenix Mutual, Hartford, Conn.
George Goodkin, M. D.	Equitable Life Assurance, New York City
Harold M. Goodman, M. D.	Home Beneficial, Richmond, Va.
Harry W. Goos, M. D.	Home, Philadelphia, Pa.
J. Keith Gordon, M. D.	Sun, Montreal, Canada
Robert S. Gordon, M. D.	Security-Connecticut, New Haven, Conn.
Charles D. Gossage, M. D.	Confederation, Toronto, Canada
Angus S. Graham, M. D.	London Life, London, Canada
George M. Graham, M. D.	Lincoln National, Fort Wayne, Ind.
Albert L. Grasmick, M. D.	Equitable Life Assurance, New York City
Ghent Graves, M. D.	American General, Houston, Tex.
Harris M. Gray, M. D.	Manufacturers, Toronto, Canada
Floyd M. Green, M. D.	Columbus Mutual, Columbus, Ohio
Bruce F. Grotts, M. D.	Prudential, Chicago, Ill.
Albert C. Grunow, M. D.	Prudential, Newark, N. J.

Richard S. Gubner, M. D.	Equitable Life Assurance, New York City
James R. Gudger, M. D.	Mutual, New York City
Van W. Gunter, M. D.	Jefferson Standard, Greensboro, N. C.
Frank R. N. Gurd, Ph. D.	Equitable Life Assurance, New York City
Milton W. Gwinner, M. D.	Western and Southern, Cincinnati, Ohio
Thomas R. Hale, M. D.	Sun, Montreal, Canada
I. Macdonald Hall, M. D.	Massachusetts Mutual, Springfield, Mass.
Llewellyn Hall, M. D.	Phoenix Mutual, Hartford, Conn.
F. Tulley Hallam, M. D.	Bankers, Des Moines, Iowa
John H. Halliday, M. D.	Australian Mutual, Sydney Australia
Gerald W. Halpenny, M. D.	Royal, Montreal, Canada
Vincent G. Hammond, M. D.	Security Mutual, Binghamton, N. Y.
Ottis E. Hanes, M. D.	Life Ins. Co. of Ga., Atlanta, Ga.
John A. A. Harcourt, M. D.	Toronto Mutual, Toronto, Canada
Frank F. Harris, M. D.	Volunteer State, Chattanooga, Tenn.
Keith S. Harrison, M. D.	Australian Mutual, Sydney, Australia
Albert H. Harrop, M. D.	Great-West, Winnipeg, Can.
T. Haynes Harvill, M. D.	Preferred Life, Dallas, Tex.
Garland M. Harwood, M. D.	Life Insurance Co. of Virginia, Richmond, Va.
Louis E. Hathaway, Jr., M. D.	Monarch, Springfield, Mass.
Howard L. Hauge, M. D.	New York Life, New York City
Walter C. Hausheer, M. D.	Prudential, Newark, N. J.

Harry M. Hawkins, M. D.	Old Line, Milwaukee, Wis.
Thomas L. Hawkins, M. D.	Western, Helena, Mont.
J. Harry Hayes, M. D.	Union, Little Rock, Ark.
Harvey D. Hebb, M. D.	Life Insurance Co. of Alberta Edmonton, Canada
Milton H. Henderson, M. D.	Excelsior, Toronto, Canada
Olin C. Hendrix, M. D.	New England Mutual, Boston, Mass.
Roger E. Henning, M. D.	Aid Association for Lutherans, Appleton, Wis.
William A. Henry, M. D.	Franklin, Springfield, Ill.
Ivan C. Heron, M. D.	West Coast, San Francisco, Calif.
Edwin J. Herpich, M. D.	National American, Baton Rouge, La.
Harry H. Hershey, M. D.	Equitable Life Assurance, New York City
William D. Hickerson, M. D.	Union Central, Cincinnati, Ohio
Eugene V. Higgins, M. D.	North American Reassurance, New York City
William H. Hill, M. D.	Progressive Life, Atlanta, Ga.
Ernest C. Hillman, Jr., M. D.	Mutual Benefit, Newark, N. J.
Harold O. Hofmeyer, M. D.	Union Guarantee, Cape Town, South Africa
Joseph C. Horan, M. D.	Metropolitan, New York City
Arnold B. Houston, M. D.	Great-West, Winnipeg, Canada
Edward G. Howe, M. D.	Prudential, Newark, N. J.
Thomas B. Hoxie, M. D.	New York Life, New York City
Henry W. Hudson, M. D.	Loyal Protective, Boston, Mass.
Gene I. Hull, M. D.	Bankers, Des Moines, Iowa
Merwin L. Hummel, M. D.	Acacia Mutual, Washington, D. C.

Arthur A. Humphrey, M. D.	Federal Life and Casualty, Battle Creek, Mich.
John L. Humphreys, M. D.	Lincoln National, Ft. Wayne, Ind.
James H. Humphries, M. D.	Home, New York City
J. Edward Hunsinger, M. D.	Republic Nat'l, Dallas, Tex.
Benjamin L. Huntington, M. D.	John Hancock Mutual, Boston, Mass.
John J. Hutchinson, M. D.	New York Life, New York City
J. Raymond B. Hutchinson, M. D.	Acacia Mutual, Washington, D. C.
Albert S. Irving, M. D.	Commonwealth, Louisville, Ky.
J. Grant Irving, M. D.	Aetna, Hartford, Conn.
Tsugitake Isshiki, M. D.	Asahi Mutual, Tokyo, Japan
Samuel Jagoda, M. D.	State Reserve, Fort Worth, Tex.
Fred E. Jenkins, M. D.	Equitable Life Assurance, New York City
Hubert R. John, M. D.	Maccabees, Detroit, Mich.
Joseph W. Johnson, Jr., M. D.	Interstate Life and Accident, Chattanooga, Tenn.
Nathaniel Jones, M. D.	Peninsular Life, Jacksonville, Fla.
Alfred Kahn, Jr., M. D.	National Equity, Little Rock, Ark.
Walter J. Karr, M. D.	Benefit Asso. Railway Employees, Chicago, Ill.
Victor L. Karren, M. D.	Home, New York City
Edward A. Keenleyside, M. D.	Prudential, Toronto, Canada
Frank J. Kefferstan, II, M. D.	John Hancock Mutual, Boston, Mass.

Charles H. Kelley, M. D.	Columbian National, Boston, Mass.
Newell R. Kelley, M. D.	Bankers, Des Moines, Iowa
Robert D. Ketchum, M. D.	Prudential, Los Angeles, Calif.
Roeinton B. Khambatta, M. D.	American Life, Karachi, Pakistan
Norvin C. Kiefer, M. D.	Equitable Life Assurance, New York City
Charles E. Kiessling, M. D.	Prudential, Newark, N. J.
Donald G. Kilgore, M. D.	Republic National, Dallas, Tex.
Glenn J. Kimball, M. D.	Paul Revere, Worcester, Mass.
Richard King, M. D.	Family Fund, Atlanta, Ga.
Carl T. Kirchmaier, M. D.	Life & Casualty, Nashville, Tenn.
Henry B. Kirkland, M. D.	Prudential, Newark, N. J.
Norman L. Knott, M. D.	Prudential, Los Angeles, Calif.
Edward Kuck, M. D.	Union Central, Cincinnati, Ohio
Paul Kurzweg, Jr., M. D.	All American Assurance, Lafayette, La.
Ewart R. Lamb, M. D.	Paul Revere, Hamilton, Canada
Phillips Lambkin, M. D.	Guardian, New York City
Paul H. Langner, Jr., M. D.	Provident Mutual, Philadelphia, Pa.
L. Gordon LaPointe, M. D.	Manhattan Life, New York City
H. Franklyn Laramore, M. D.	Connecticut Mutual, Hartford, Conn.
Rodney C. Larcom, Jr., M. D.	Massachusetts Indemnity, Boston, Mass.
Walter F. Larrabee, Jr., M. D.	Minnesota Mutual, St. Paul, Minn.
Albert L. Larson, M. D.	Travelers, Hartford, Conn.

Dwight Lawson, M. D.	Victory, Topeka, Kan.
C. Marshall Lee, Jr., M. D.	John Hancock, Boston, Mass.
James M. Leffel, M. D.	Empire L. & A., Indianapolis Ind.
Harold R. Leffingwell, M. D.	Paul Revere, Worcester, Mass.
Henry J. Lehnhoff, Jr., M. D.	Woodmen of the World, Omaha, Neb.
Richard J. Lempke, M. D.	Mutual, New York City
Clark H. Lentz, M. D.	Business Men's, Kansas City, Mo.
Charles P. LeRoyer, Jr., M. D.	Travelers, Hartford, Conn.
William R. Leute, Jr., M. D.	Penn Mutual, Philadelphia, Pa.
Janus C. Lindner, M. D.	Ohio National, Cincinnati, Ohio
Everett H. Lindstrom, M. D.	Western, Helena, Mont.
Roger P. Lochhead, M. D.	Bankers National, Montclair, N. J.
Gladstone W. Lougheed, M. D.	Confederation, Toronto, Canada
John F. Lovejoy, M. D.	United Life, Jacksonville, Fla.
David Luchs, M. D.	Connecticut Mutual, Hartford, Conn.
Gerald J. Lunz, M. D.	Knights of Columbus, New Haven, Conn.
Frank M. McChesney, M. D.	Equitable, Washington, D. C.
William J. McCristal, M. D.	City Mutual, Sydney, Australia
Howard M. McCue, Jr., M. D.	Life Insurance Co. of Virginia, Richmond, Va.
Murdo G. MacDonald, M. D.	National, Montpelier, Vt.
William MacDonald, M. D.	Teachers Insurance & Annuity Association, New York City

Duncan T. McEwan, M. D.	National Standard, Orlando, Fla.
Arthur J. McGanity, M. D.	Dominion, Waterloo, Canada
J. David McGaughey, III, M. D.	Connecticut General, Hartford, Conn.
Frank J. McGurl, M. D.	Prudential, Houston, Tex.
Thomas J. McGurl, Jr., M. D.	Mutual, New York City
J. Stewart McInnes, M. D.	Sovereign, Winnipeg, Can.
Charles D. McKeown, M. D.	Farmers & Bankers, Wichita, Kan.
George McLean, M. D.	Sun, Baltimore, Md.
Lawrence L. McLellan, M. D.	Provident Mutual, Philadelphia, Pa.
William J. McNamara, M. D.	Equitable Life Assurance, New York City
Charles D. Magee, M. D.	Western and Southern, St. Louis, Mo.
Morton Magiday, M. D.	Equitable Life Assurance, New York City
S. J. Newton Magwood, M. D.	Continental, Toronto, Canada
John Malgieri, M. D.	New York Life, New York City
Arthur F. Mangelsdorff, M. D.	Prudential, Newark, N. J.
J. Herbert Marks, M. D.	Guaranty Union, Beverly Hills, Calif.
Clement G. Martin, M. D.	Continental Assurance, Chicago, Ill.
Peter V. Martin, M. D.	Prudential, Minneapolis, Minn.
Francis A. L. Mathewson, M. D.	Great-West, Winnipeg, Canada
Ferald D. Mauk, M. D.	Republic National, Dallas, Texas
John W. Merritt, M. D.	Dominion, Waterloo, Canada
Ignacio Mesa, M. D.	"La Latino-Americana", Mexico City, Mexico
Paul S. Metzger, M. D.	Nationwide Life, Columbus, Ohio

Lloyd C. Miller, M. D.	National Life & Accident, Nashville, Tenn.
Milton B. Miller, M. D.	Victory Life, Topeka, Kan.
Edward S. Mills, M. D.	Prudential Assurance, Montreal, Canada
Ian G. Milne, M. D.	Prudential Assurance, Montreal, Can.
Gerald S. Modjeska, M. D.	Continental Assurance, Chicago, Ill.
Eugene Montgomery, M. D.	North American, Toronto, Canada
Richard C. Montgomery, M. D.	Manufacturers, Toronto, Canada
John F. Moore, Jr., M. D.	Mutual, New York City
Samuel R. Moore, M. D.	Life of North America, Philadelphia, Pa.
J. R. E. Morden, M. D.	Massachusetts Mutual, Springfield, Mass.
J. Palmer Moss, M. D.	Columbian Mutual, Memphis, Tenn.
Bernard Mount, M. D.	Southern United, Montgomery, Ala.
Elmer B. Mountain, M. D.	American Mutual, Des Moines, Iowa
Clifford V. Mulligan, M. D.	T. Eaton, Toronto, Canada
Luiz Murgel, M. D.	Companhia Internacional, Rio de Janeiro, Brazil
George H. Murphy, M. D.	Maritime, Halifax, Canada
John R. Murphy, M. D.	North American Reassurance, New York City
Sidney A. Narins, M. D.	Mutual, New York City
Richard M. Nay, M. D.	Indianapolis Life, Indianapolis, Ind.
John B. Neal, M. D.	Prudential, Jacksonville, Fla.
Mather H. Neill, M. D.	Aetna, Hartford, Conn.
Richard A. Nelson, M. D.	Prudential, Jacksonville, Fla.

Richard E. Nicholson, M. D.	Connecticut Mutual, Hartford, Conn.
Charles A. Nordin, M. D.	Equitable, Des Moines, Iowa
William P. Nuessle, M. D.	Prudential, Minneapolis, Minn.
Andrew J. Oberlander, M. D.	Prudential, Chicago, Ill.
William L. O'Connell, M. D.	Union Labor, New York City
Robert D. O'Connor, M. D.	Old Line, Milwaukee, Wis.
Robert J. Oehrig, M. D.	Home, New York City
Denis J. O'Leary, M. D.	New York Life, New York City
Martin I. Olsen, M. D.	Central, Des Moines, Iowa
William F. H. O'Neill, M. D.	Franklin, Springfield, Ill.
Earl T. Opstad, M. D.	Northwestern National, Minneapolis, Minn.
John K. T. Ormrod, M. D.	Aetna, Hartford, Conn.
Arthur E. Parks, M. D.	Canada Life, Toronto, Canada
John S. Pearson, M. D.	American United, Indianapolis, Ind.
John McC. Peck, M. D.	John Hancock Mutual, Boston, Mass.
D. Sergeant Pepper, M. D.	Connecticut Mutual, Hartford, Conn.
Gilberto S. Pesquera, M. D.	Metropolitan, New York City
Charles A. Peters, M. D.	Prudential Assurance, Montreal, Canada
Ray W. Peterson, M. D.	Columbian Mutual, Binghamton, N. Y.
Frank I. Pitkin, M. D.	New England Mutual, Boston, Mass.
Theodore E. Plucinski, M. D.	Mutual, New York City

Albert A. Pollack, M. D.	Mutual, New York City
Fred F. Porter, M. D.	Prudential, Chicago, Ill.
Herbert E. Pugsley, M. D.	Confederation, Toronto, Canada
William O. Purdy, M. D.	Equitable, Des Moines, Iowa
Raymond S. A. Purkis, M. D.	Canada Life, Toronto, Canada
Michael A. Puzak, M. D.	Peoples, Washington, D. C.
Paul M. Rattan, M. D.	Great National, Dallas, Tex.
James H. Ready, M. D.	General American, St. Louis, Mo.
Clifton L. Reeder, M. D.	Continental Assurance, Chicago, Ill.
Paul V. Reinartz, M. D.	Prudential, Jacksonville, Fla.
Whitman M. Reynolds, M. D.	Equitable Life Assurance, New York City
H. Guy Riche, M. D.	Guaranty Income, Baton Rouge, La.
Albert V. Rigsbee, M. D.	Acacia Mutual, Washington, D. C.
Donald F. Rikkers, M. D.	Northwestern Mutual, Milwaukee, Wis.
Robert C. Roadhouse, M. D.	Prudential, Toronto, Canada
George P. Robb, M. D.	Metropolitan, New York City
David C. Roberts, M. D.	Guardian, New York City
Albert J. Robinson, M. D.	Connecticut General, Hartford, Conn.
John C. Robinson, M. D.	Travelers, Hartford, Conn.
Van C. Robinson, M. D.	American Mutual, Des Moines, Iowa
Franklin N. Roemhild, M. D.	Prudential, Houston, Tex.
Wieland W. Rogers, M. D.	Prudential, Jacksonville, Fla.

Henry B. Rollins, M. D.	Connecticut Mutual, Hartford, Conn.
Alberto Z. Romualdez, M. D.	National Life, Manila, P. I.
Erle S. Ross, M. D.	Brotherhood of Railroad Trainmen, Cleveland, Ohio
John G. Ross, M. D.	Mutual, Waterloo, Canada
Thomas F. Ross, M. D.	Ohio State, Columbus, Ohio
Edward W. Rowe, M. D.	Midwest, Lincoln, Neb.
Bartholomew A. Ruggieri, M. D.	Bankers National, Montclair, N. J.
John K. Ruggles, Jr., M. D.	Paul Revere, Worcester, Mass.
Norman R. Ruud, M. D.	Phoenix Mutual, Hartford, Conn.
Merlin T. Ryman, M. D.	Mutual Benefit, Newark, N. J.
Dan Y. Sage, M. D.	Southern, Atlanta, Ga.
John L. Saia, M. D.	National, Montpelier, Vt.
Joe H. Sanderlin, M. D.	First Pyramid Life, Little Rock, Ark.
Raymond C. Scannell, M. D.	Security Life and Accident, Denver, Colo.
Royal S. Schaaf, M. D.	Prudential, Newark, N. J.
Kenneth F. Schaefer, M. D.	Prudential, Minneapolis, Minn.
Frederick L. Scheyer, M. D.	Family Life, Seattle, Wash.
Robert B. Schlesinger, M. D.	Mutual Trust, Chicago, Ill.
Paul G. Schwager, M. D.	Equitable, Waterloo, Canada
Berthold T. D. Schwarz, M. D.	Bankers National, Montclair, N. J.
William H. Scoins, M. D.	Lincoln National, Ft. Wayne, Ind.
Robert J. Scott, M. D.	Michigan Life, Detroit, Mich.

Ralph C. Secor, M. D.	Liberty National, Birmingham, Ala.
Alfred F. Seibert, M. D.	Travelers, Hartford, Conn.
David L. Selby, M. D.	Imperial, Toronto, Canada
Thomas S. Sexton, M. D.	Massachusetts Mutual, Springfield, Mass.
Hall Shannon, M. D.	Southland, Dallas, Tex.
Elroy F. Sheldon, M. D.	Occidental, Los Angeles, Calif.
Joyce T. Sheridan, M. D.	Fidelity Mutual, Philadephia, Pa.
Hubert H. Shook, M. D.	Ohio National, Cincinnati, Ohio
Vaughan P. Simmons, M. D.	Northwestern Mutual, Milwaukee, Wis.
William H. Simmons, M. D.	Travelers, Hartford, Conn.
George A. Simpson, M. D.	Colonial Life, East Orange, N. J.
Howard N. Simpson, M. D.	Monarch Life, Springfield, Mass.
Richard B. Singer, M. D.	New England Mutual, Boston, Mass.
James R. Slamer, M. D.	Northwestern Mutual, Milwaukee, Wis.
Noah H. Sloan, M. D.	Allstate Life, Skokie, Ill.
John T. Smiley, M. D.	Prudential, Minneapolis, Minn.
F. Hartley Smith, M. D.	Great-West, Winnipeg, Canada
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Pacific Mutual Life Ins. Co., Los Angeles, Calif.	F. R. Anderson, M. D. J. C. Talbot, M. D. R. J. Walker, M. D.
Pan-American Life Ins. Co., New Orleans, La.	R. C. Voss, M. D.
Paul Revere Life Ins. Co., Worcester, Mass.	G. J. Kimball, M. D. E. R. Lamb, M. D. H. R. Leffingwell, M. D. J. K. Ruggles, Jr., M. D.
Pearl Assurance Co., Ltd., Toronto, Canada	A. W. Capon, M. D.
Peninsular Life Insurance Co., Jacksonville, Fla.	Nathaniel Jones, M. D.
Penn Mutual Life Ins. Co., Philadelphia, Pa.	J. R. Bowen, M. D. B. A. Dawber, M. D. W. R. Leute, Jr., M. D. R. L. Weaver, M. D.
Peoples Life Insurance Co., Frankfort, Ind.	C. A. Burroughs, M. D.
Peoples Life Insurance Co., Washington, D. C.	M. A. Puzak, M. D.
Philadelphia Life Ins. Co., Philadelphia, Pa.	T. M. Armstrong, M. D. D. B. Gelfond, M. D. P. V. Martin, M. D.
Philippine American Life Ins. Co., Manila, P. I.	F. P. Valenzuela, M. D.
Phoenix Mutual Life Ins. Co., Hartford, Conn.	W. R. Bradley, M. D. R. A. Goodell, M. D. Llewellyn Hall, M. D.

Pilot Life Insurance Co., Greensboro, N. C.	J. L. Cook, M. D. H. F. Starr, Jr., M. D.
Pioneer American Insurance Co., Fort Worth, Tex.	W. S. Barcus, M. D.
Plymouth Mutual Life Insurance Co., Philadelphia, Pa.	M. W. Fischbach, M. D.
Postal Life Insurance Co., New York City	L. B. Dunn, M. D.
Preferred Life Assurance Society, Montgomery, Ala.	B. C. Bird, M. D.
Preferred Life Insurance Co., Dallas, Tex.	T. H. Harvill, M. D.
Progressive Life Insurance Co., Atlanta, Ga.	W. H. Hill, M. D.
Protective Life Insurance Co., Birmingham, Ala.	E. G. Givhan, Jr., M. D.
Provident Indemnity Life Insurance Co., Norristown, Pa.	L. F. Flick, M. D.
Provident Life and Accident Ins. Co., Chattanooga, Tenn.	W. R. Bishop, M. D.
Provident Mutual Life Ins. Co., Philadelphia, Pa.	E. J. Brogan, M. D. P. H. Langner, Jr., M. D. L. L. McLellan, M. D.
Prudential Assur. Co., Ltd., Montreal, Canada	D. G. Cameron, M. D. E. S. Mills, M. D. I. A. Milne, M. D.

Prudential Insurance Co. of America, Newark, N. J.	S. F. Bassett, M. D. R. D. Brewer, Jr., M. D. R. F. Buchan, M. D. R. H. Craig, M. D. A. H. Domm, M. D. R. M. Donauer, M. D. V. J. Donnelly, M. D. R. L. Dross, M. D. R. E. Funke, M. D. F. I. Ganot, M. D. B. F. Grotts, M. D. A. C. Grunow, M. D. W. C. Hausheer, M. D. E. G. Howe, M. D. E. A. Keenleyside, M. D. R. D. Ketchum, M. D. C. E. Kiessling, M. D. H. B. Kirkland, M. D. N. L. Knott, M. D. A. F. Mangelsdorff, M. D. F. J. McGurl, M. D. J. B. Neal, M. D. R. A. Nelson, M. D. W. P. Nuessle, M. D. A. J. Oberlander, M. D. F. F. Porter, M. D. P. V. Reinartz, M. D. R. C. Roadhouse, M. D. F. H. Roemhild, M. D. W. W. Rogers, M. D. R. S. Schaaf, M. D. K. F. Schaefer, M. D. J. T. Smiley, M. D. R. A. Snider, M. D.
Puritan Life Insurance Co., Providence, R. I.	J. A. Dillon, M. D.
Reliance Mutual Life Insurance Co., Park Ridge, Ill.	S. S. Werth, M. D.
Republic National Life Ins. Co., Dallas, Tex.	J. E. Hunsinger, M. D. D. G. Kilgore, M. D. F. D. Mauk, M. D.
Reserve Life Insurance Co., Dallas, Tex.	D. W. Carter, Jr., M. D.

Rockford Life Insurance Co., Rockford, Ill.	P. A. Anderson, M. D.
Royal Insurance Co., Ltd., Montreal, Canada	G. W. Halpenny, M. D.
Security Benefit Life Ins. Co., Topeka, Kan.	F. R. Stearns, M. D.
Security-Connecticut Life Ins. Co., New Haven, Conn.	R. S. Gordon, M. D.
Security Life and Accident Co., Denver, Colo.	R. C. Scannell, M. D.
Security Life & Trust Co., Winston-Salem, N. C.	J. P. Davis, M. D.
Security Mutual Life Ins. Co., Binghamton, N. Y.	V. G. Hammond, M. D.
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Southland Life Insurance Co., Dallas, Tex.	Hall Shannon, M. D.
Southwestern Life Ins. Co., Dallas, Tex.	C. F. Brown, M. D. C. E. Cook, M. D.
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Standard Insurance Company, Portland, Ore.	E. L. Boylen, M. D.
Standard Life Association, Lawrence, Kan.	G. D. Townshend, M. D.
Standard Life Assur. Co., Montreal, Canada	W. W. Eakin, M. D.

State Farm Life Insurance Co., Bloomington, Ill.	J. T. France, M. D. E. M. Stevenson, M. D.
State Mutual Life Assur. Co., Worcester, Mass.	F. P. Bicknell, M. D. R. L. Candage, M. D.
State Reserve Life Insurance Co., Fort Worth, Tex.	Samuel Jagoda, M. D.
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T. Eaton Life Assurance Co., Toronto, Canada	C. V. Mulligan, M. D.
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Texas Prudential Insurance Co., Galveston, Tex.	E. R. Thompson, M. D.
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Victory Mutual Life Insurance Co., Chicago, Ill.	A. C. Albright, M. D.
Volunteer State Life Ins. Co., Chattanooga, Tenn.	F. F. Harris, M. D.
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